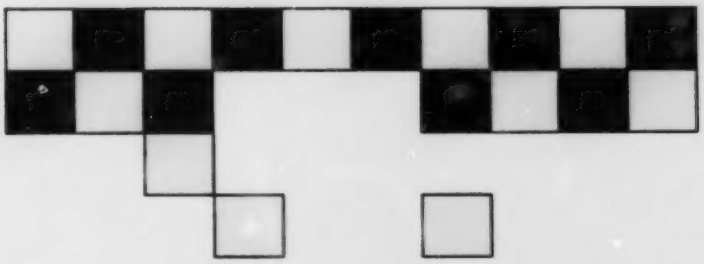
CANADIAN GUIDELINES FOR HEALTHY WEIGHTS

Report of an Expert Group
convened by
Health Promotion Directorate
Health Services and Promotion Branch



Health and Welfare Canada
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CANADIAN GUIDELINES FOR HEALTHY WEIGHTS

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PREFACE

The creation of Canadian guidelines for healthy weights is essential to a public health strategy promoting healthy weights. This publication reviews existing weight measures and the epidemiological evidence leading to the selection of appropriate guidelines for assessing weight in relation to health.

Canadian Guidelines for Healthy Weights was developed by an Expert Group on Weight Standards, and reviewed by over fifty experts in the field of weight assessment. We encourage the widespread acceptance of their recommendations.

We can benefit from your comments on your experiences in applying these new Canadian guidelines for healthy weights. They will assist us in developing future documents relating to these guidelines.

Please address your comments to:

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I. EXECUTIVE SUMMARY

Expert Group on Weight Standards

This report on Canadian Guidelines for Healthy Weights was prepared by Marielle Ledoux, Ph.D., Chairman, Expert Group on Weight Standards for submission to the Minister of the Department of National Health and Welfare on March 31, 1987. Members of the Expert Group participated in three meetings, reviewed documents, prepared reports as required and undertook consultative activities in the development of the report. Recommendations for the implementation of the report through publication in suitable formats for both the general public and health professionals were made.

Purpose of Weight Guidelines

In Canada, guidelines which associate weight and health for Canadians have never been developed. Consequently, there are no Canadian weight guidelines which have been accepted nationally as a basis for goal-setting for professionals, policy makers and the general public. Both the public and professionals continue to indicate a need for a reliable tool which can be easily used for the assessment of weight status.

There is a need to reach agreement on the use of one set of guidelines which could be used to determine an acceptable range of healthy weights for Canadians and to identify conditions of excess weight and underweight. The recognition of an acceptable range of healthy weights is fundamental to responding to the increased preoccupation with weight and the explorative practices which often result. An acceptable range of healthy weights can also help Canadians understand that normal fluctuations in weight can occur.

There is a need to use Canadian data as a reference point for weight guidelines because each population should have unique guidelines based on its own morbidity/mortality experiences. Data from two major Canadian studies, the 1981 Canada Fitness Survey and the 1978 Canada Health Survey are available for analysis of the relationship of weight to health.

Because of gaps in the Canadian literature, there is also a need to establish the relationship of weight to mortality and morbidity rates by reviewing major international prospective and retrospective studies.

The development of new weight guidelines for Canadians is consistent with an overall objective for national nutrition programs which is to increase the proportion of Canadians who adopt nutrition practices consistent with attaining and maintaining appropriate body weights.

The overall purposes set by the Expert Group in developing Canadian weight-health guidelines to be used by both health professionals and the general public were:

1. to provide a scientifically based assessment tool which will also be simple and practical

2. to identify patterns of weight including acceptance of excess and low weights as they relate to health risks or clinical problems

3. to establish appropriate, realistic weights which relate to positive body image.

Selection of Guidelines

The Expert Group on Weight Standards was asked to select a measure which would reflect the relationship between weight and health. The measure selected would not be intended for use to assess either body composition or physical fitness.

The criteria selected by the Expert Group for the "best measure" of weight in relation to morbidity and mortality data are: epidemiological validity, precision, reliability, accuracy, skill, availability, cost and client acceptability. These criteria were applied against common measurements used in prospective and retrospective studies including the two major Canadian studies.

The recommendations of the Expert Group are for the public to use four zones of the Body

Mass Index (BMI) as a measure for healthy weights, and for health professionals to also use Waist-Hip Ratio measurements as indicators of fat distribution.

Body Mass Index (BMI)

The Body Mass Index (BMI), which is defined as weight (kg) divided by height (metres) squared (Wt/Ht^2), was selected as the primary weight measurement as it met all of the proposed criteria. The BMI is a measure which is epidemiologically valid for public health purposes from the perspective of determining high-risk groups. The BMI may also be used by the general public as a measure which describes the chance of developing specific health problems considered in the development of the guidelines.

The cut-off points chosen were:

Zone A BMI less than 20

This zone is defined as the zone that "may be associated with health problems for some people".

Note: Entry into this zone should be used as a trigger for any underlying cause of the low BMI.

*Zone B BMI between 20 and 25

This zone is defined as the zone associated with "good weight for most people".

Note: This zone is associated with the lowest occurrence of morbidity and mortality.

*Zone C BMI between 25 and 27

This zone is defined as the zone that "may lead to health problems in some people".

Note: Because sharp increases in morbidity and mortality have been reported for a BMI around 27, a caution zone is suggested.

Zone D BMI greater than 27

This zone is defined as the zone associated with "increasing risk of developing health problems".

Note: Studies show that BMIs greater than 27 are associated with an increased occurrence of hypertension, hyperlipidemia and coronary heart disease.

* These two zones represent the "generally acceptable range" of weight for health.

Interpretation:

1. The Body Mass Index (BMI) depends upon measures of weight and height. Both these measures are easily accessible and highly accurate.

2. The BMI scale should be used as a "continuum" where the risk of developing health problems increases with shifts away from the "generally acceptable range".

3. Rapid changes in weight within and between zones should be considered as important indicators of potential problems particularly at the outer ends of the scale.

4. The BMI has been used primarily to identify high-risk groups in a specific population. Interpretation of the BMI for factors such as age and duration of weight problems is essential in applying this measure to individuals.

5. Because of variations in the association of BMI to risk factors and/or specific diseases, it is important to consider the fact that the choice of cut-off points represents the best choice to date based on data available at the present time. Young males with a BMI of 27 and over should be particularly concerned about an increased probability of developing health problems.

6. The same cut-off points for the Body Mass Index $Wt(kg)/Ht^2(m)$ are appropriate for

both men and women 20 to 65 years of age.

7. In the Canadian Guidelines for Healthy Weights, BMI is being used to reflect weight in relation to health. However, the BMI alone is not a direct measure of body fatness. It should be recognized that a BMI above the "generally acceptable range" of weight for health is not necessarily indicative of excess fat. A short, stocky muscular individual, for example, may have a BMI above 27, yet not be at risk for his/her health. At the same time, a BMI below the "generally acceptable range" of weight for health is not necessarily indicative of insufficient body fat and increased risk. For example, healthy endurance athletes may maintain a BMI below the "generally acceptable range" for years. Interpretation of BMI values in these cases should be made with caution.

Waist-Hip Ratio (WHR)

Fat distribution may be a better indicator of the presence of risk factors (hypertension, hyperlipidemia) or a better predictor of some diseases (coronary heart disease, diabetes) than the relation of weight to height. Therefore, measures of fat distribution should also be considered in the development of new guidelines.

The Waist-Hip Ratio, which is the waist circumference divided by the hip circumference, meets most of the criteria set by the Expert Group. It is suggested that it be included as an indicator of fat distribution in view of its impact on cardiovascular risk factors. However, further research is necessary before a detailed recommendation can be made. The values suggested by Björntorp (1985) are proposed for interim use: WHR value of 1.0 for men and 0.8 for women. Values in excess of these may be associated with increasing health risks.

Interpretation:

1. Waist-Hip Ratio can be used by health professionals as an indicator of fat distribution.

2. No scale of measurement or "continuum" showing the increasing probability of developing health problems is available for the Waist-Hip Ratio at this time.

3. The values suggested by Björntorp (1985) for the Waist-Hip Ratio can be used as indicative of increased risk. However, further research is necessary before detailed recommendations on its use can be made.

4. It is possible to change one's body composition through diet and/or regular physical activity. This may result in a loss of fat, a slight increase in muscle mass and a trimmer figure with little or no change in body weight. So even if a BMI remains stable, a favourable change in the WHR may signal reduced risk for an individual.

Weight for Health Status of Canadians

The weight guidelines proposed by the Expert Group on Weight Standards were applied to the 1981 Canada Fitness Survey, the most recent national study which included anthropometric measurements.

Highlights:

1. One out of every two adult Canadians between 20 and 69 years of age was at a "good weight" for health (Zone B).

2. More men (29%) than women (19%) were at "increasing risk of developing health problems" (Zone D) because of excess weight.

3. More women (15%) than men (5%) were in a category which "may be associated with health problems in some people" (Zone A) because of underweight.

4. For both men and women, there was an "increasing risk of developing health problems associated with weight" (Zone D) with increasing age.

II. INTRODUCTION

Deciding on a weight which is compatible with health is important for both health professionals and for individuals in promoting health and preventing and treating disease.

Over the past ten years, there has been an increasing awareness that measures, such as height-weight tables, and skinfold measurements, require reassessment in light of more recent scientific, clinical and epidemiological evidence.

At the same time, changing concepts about weight for health have emerged which suggest that the overriding concern with excess weight should be considered along with problems of underweight.

An Expert Group on Weight Standards under the chairmanship of Marielle Ledoux, Ph.D., was invited by the Health Promotion Directorate, Health and Welfare Canada, to undertake an examination of weight guidelines for Canadians.

The mandate of the Expert Group was:

1. to make specific recommendations to the Health Promotion Directorate on:

   a) weight standards for healthy Canadian adults (20 to 65 years of age)

   b) the content and format for presentation of weight standards to the public and health professionals

2. to establish a reasonable healthy weight in numerical terms for the Canadian population

3. to prepare and present a report containing:

   a) the recommendations

   b) the rationale and scientific basis for selection of the standards. A description of the relevance of weight control to health status e.g. risk factors or diseases such as coronary heart disease, cancer and diabetes throughout the life cycle is to be included.

Following discussion by the Expert Group, consensus was reached on the expansion of project goals to include:

4. to obtain standards which health professionals can use in the assessment of the health status of their patients.

5. to develop weight standards or other means that will serve as a tool for counselling by health professionals and policy makers to establish realistic weight goals and to adopt appropriate intervention strategies.

Members of the Expert Group on Weight Standards were:

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III. DEVELOPING WEIGHT GUIDELINES FOR CANADIANS

1.0 Purpose of Weight Guidelines

In Canada, guidelines which associate weight and health for Canadians have never been developed. Consequently, there are no Canadian weight guidelines which have been nationally accepted as a basis for goal-setting for professionals, policy makers and the general public. At this time, both the public and professionals continue to indicate a need for a reliable tool which can be easily used for assessment of weight.

There is a need to reach agreement on the use of one set of guidelines which could be used to determine an acceptable range of healthy weights for Canadians and to identify conditions of excess weight and underweight. The recognition of an acceptable range of healthy weights is fundamental to responding to the increased preoccupation with weight and the exploitive practices which often result. An acceptable range of healthy weights can also help Canadians understand that normal fluctuations can occur.

There is a need to use Canadian data as a reference point for weight guidelines because each population should have unique guidelines based on its own morbidity/mortality experiences. Data from two major Canadian studies, the 1981 Canada Fitness Survey and the 1978 Canada Health Survey, are available for analysis of the relation between weight and health. Because of gaps in the Canadian literature, there is also a need to establish the relation between weight and mortality and morbidity rates by reviewing major international prospective and retrospective studies.

The development of new weight guidelines for Canadians is consistent with an overall objective for national nutrition programs which is to increase the proportion of Canadians who adopt nutrition practices consistent with attaining and maintaining appropriate body weights.

The overall purposes set by the Expert Group in developing Canadian weight-health guidelines to be used by both the health professionals and the general public were:

1. to provide a scientifically based assessment tool which will also be simple and practical

2. to identify patterns of weight including acceptable excess and low weights as they relate to health risks or clinical problems

3. to establish appropriate, realistic weights which relate to positive body image.

2.0 Activities Related to Weight and Weight Guidelines

The development of weight guidelines is closely linked to two other activities currently under way in the Department of National Health and Welfare.

One project is the work of the Task Force on the Treatment of Obesity supported by the Health Services Directorate, Health Services and Promotion Branch. The Task Force's report is intended to support guidelines on the treatment of obesity. There has been close collaboration between the Expert Group on Weight Standards and the Task Force on the Treatment of Obesity to ensure consistency.

A second project is the development of a health promotion strategy for weight under the mandate of the Health Promotion Directorate, Health Services and Promotion Branch. There was collaboration between the chairman of the Expert Group on Weight Standards and the program officers in the development of the paper "Promoting Healthy Weights: A Discussion Paper".

3.0 Process in the Development of Consensus

Members of the Expert Group participated in three meetings, reviewed documents, prepared reports as required and were involved in consultative activities.

The first meeting (May 10, 1985) was held:

1. to orient members to the task
2. to determine the work plan (to arrive at a final consensus)
3. to review four reports prepared by Dr. White and collaborators which are:
   - Stockwell, H. and F. White. Application of existing Canadian data bases to the epidemiological study of under/overweight in Canada in reference to the development of a health promotion strategy. Dalhousie University, Halifax, December 14, 1984.
   - Stockwell, H. and L. Periera, F. White and C. Cottereau. The association between body weight and leading causes of morbidity and mortality: an epidemiological reconnaissance. Dalhousie University, Halifax, February 20, 1984.
   - White, F. and L. Periera. Measurements of obesity and overweight in practical settings: an epidemiological perspective. Dalhousie University, Halifax, February 28, 1985.
   - White, F. The epidemiology of overweight in Canada in reference to the development of a health promotion strategy. Summary report. Dalhousie University, Halifax, April 26, 1985.
4. to discuss with Dr. Petrasovits the main conclusions of the National Institutes of Health Consensus Development Conference on the "Health Implications of Obesity" held February 11-13, 1985
5. to review documents obtained from consultations with Dr. Castelli, Framingham, Mass. and Mr. Seltzer of Metropolitan Life Insurance Co., New York
6. to define criteria for the choice of weight measurements
7. to establish a need for a review of literature and the compilation of background documents on: epidemiological data on weight related to health (morbidity and mortality); evaluation of methods presently used with regard to weight measures; identification of data sources and further analysis focusing mainly on Canadian data; identification of gaps and limitations of weight measures.

Following the first meeting, reviews were requested from Dr. White and Dr. Roncari. The three documents which resulted from these reviews are:

- Periera, L. and F. White. Epidemiological tabulations of the body mass index in association with selected morbidity and blood chemistry indicators by age, sex and region. Volume I. Dalhousie University, Halifax. December 19, 1985 (Appendix I of this report).
- Periera, L. and F. White. Epidemiological tabulations of the body mass index in association with selected morbidity and blood chemistry indicators by age, sex and region. Volume II. Dalhousie University, Halifax. December 24, 1985 (Appendix II of this report).
- Roncari, D. Clinical-metabolic evidence for the impact of obesity on cardiovascular risk factors.

University of Calgary, Calgary. January 7, 1986 (Appendix III of this report).

The second meeting (January 22 and 23, 1986) was held:

1. to discuss the review documents prepared and presented by Dr. White and Dr. Roncari

2. to increase coordination between the Expert Group on Weight Standards, the Task Force on the Treatment of Obesity and the "Promoting Healthy Weights" activities. A meeting had been held October 10, 1985 between members of the Expert Group on Weight Standards and the Chairman and Coordinator of the Task Force Committee to identify data and research needs for both committees

3. to discuss the procedure used to assess body composition in Fitness Canada's Canadian Standardized Test of Fitness, entitled "Body Weight, Adiposity and Fat Distribution" as presented by R. Adams, Fitness Canada and Dr. Bouchard

4. to identify issues and outstanding information required to arrive at a consensus e.g. fat distribution data, limits of chosen guidelines

5. to clarify the request by the Department of National Health and Welfare for recommendations on the use and implementation of the chosen weight-health guidelines.

At the end of the second meeting, a draft statement of consensus was prepared to be presented at a meeting of the Task Force on the Treatment of Obesity. Discussion and commentaries suggested for the general public and health professionals were summarized and a chart describing the zones between the selected cut-off points was prepared.

The third meeting (August 12 and 13, 1986) or second "consensus" meeting was held:

1. to confirm the choice of cut-off points

2. to ensure coordination between the Task Force on the Treatment of Obesity and the Expert Group on Weight Standards on the choice of cut-off points

3. to determine the key items to include in the rationale for the choice of measurements and cut-off points.

Following the meeting, a first draft of the rationale was sent to all members of the Expert Group on Weight Standards, to Dr. Marliss, Chairman of the Task Force on the Treatment of Obesity, and to reviewers in the Department of National Health and Welfare. Changes were made according to suggestions forwarded to the Chairman of the Expert Group on Weight Standards. The report was finalized and submitted to the Health Promotion Directorate on March 31, 1987. Members of the Expert Group reviewed and approved the report prior to its distribution.

IV. CANADIAN GUIDELINES FOR HEALTHY WEIGHTS

1.0 Rationale for the Choice of Measurements and Cut-off Points

The development of weight guidelines involved the selection of the actual measurement(s) and the determination of appropriate cut-off points. The first step consisted of choosing measurements that would specifically express the association between weight and health. In addition, they were to be practical for individual Canadians, for those individuals or groups involved in providing assessment, counselling or education programs for the public and for those individuals or organizations involved in policy making. The setting of specific criteria and their application are described. The second step was the selection of appropriate cut-off points for the measurement(s) chosen which represent consideration of levels of risks to health. The rationale for the choice of cut-off points is described.

1.1 Criteria for the Selection of Measurements

The concept of "weight guidelines" for adults, developed in this report, has a broader meaning than solely body weight. The components to be considered in the development of new guidelines are height, body weight, body fatness and body fat distribution.

The guidelines will be used for self-assessment by the general public defined as the healthy Canadian adult population 20 to 65 years of age. Therefore, the measurement chosen should be simple to use and practical, i.e. require equipment such as scales and measuring tapes found in most Canadian households. At the same time, tables or nomograms will be needed to aid in the interpretation of the measurement for better understanding by the public. In other words, the criteria for the measurement should be: **easily available, low cost, acceptable to the client, special skills not required.**

The measurement selected will be recommended as a basis for the development of health promotion strategies including use in weight control programs and population studies to identify those at acceptable weights as well as those groups at risk. The following criteria should be applied in the assessment of measurements: **epidemiological validity, precision, reliability, accuracy** (White and Periera, 1985).

The following definitions for the criteria are (White and Periera, 1985):

Epidemiological validity "infers that a test measures what it purports to measure. In this context, validity is judged by the extent to which overweight/obesity is related to increased risk of morbidity or mortality".

Precision is the "quality of being exactly defined".

Reliability is the "quality of being always easily reproducible or repeatable".

Accuracy describes a "measurement conforming to a standard or a true value".

1.2 Advantages and Disadvantages of Most Common Measurements

Apart from the technique of densitometry which is a measurement used in research, five measurements are in common use for self-assessment and/or epidemiological studies on a prospective or retrospective basis. These measurements have been related to morbidity and/or mortality data for both men and women of various age groups. Analysis of the advantages and disadvantages of these common measurements according to criteria proposed under 1.1 was undertaken to establish the most appropriate measurement. The analysis was based on a review of the literature

including special reports by Dr. White and Dr. Roncari prepared for the Minister, Department of National Health and Welfare.

1.2.1 Weight-Height Tables

"Weight-height tables are used to describe body weight in relation to a reference population or an assumed desirable standard." (Harrison, 1985). They are simple to use, require regular household equipment and are very easy to understand. They relate weight to specific heights and/or frame size. Frame size is measured either by the wrist circumference as in 1959 Metropolitan Height and Weight Tables, (Metropolitan Life Insurance Company, 1959) or the elbow breadth (1983 Metropolitan Height and Weight Tables). Both of these measures are less affected by degree of adiposity than other anthropometric dimensions.

The terms "ideal weight" or "desirable weight" have been used to describe individual weights for height according to reference frame size. The population data used to define these guidelines are often very specific and cannot be used as "ideal standards" on an individual basis, especially if the individual is not from the same population used to develop the guidelines. For example, "standards based on insured individuals in principle should not be generalized to the general population" (White and Periera, 1985). These actuarial tables are based on a highly selective population and very obese and non-insured individuals are rejected from the sample.

The Metropolitan Height and Weight Tables (1959, 1983) which are the most widely used tables, are based on weights related to the lowest mortality. However, they do not address the issue of morbidity which is one of the major issues to be addressed in this report. Only the 1983 Metropolitan Height and Weight Tables include some discussion relating diabetes and hypertension to their suggested weights for heights. However, other diseases such as hyperlipoproteinemia and cancer, have not been discussed.

Limitations of the tables include: representation of some subjects more than once e.g. individual policies, not subjects, are tabulated; lack of measurements for body frame; some self-reported measures of height and weight; assumptions made for weight of "regular clothes" and height of shoes because individuals were not completely undressed when both measurements were taken.

Using the 1959 Metropolitan Height and Weight Tables, a relative weight index was calculated for the Framingham Study (Kannel and Gordon, 1974). At times, relative ideal weights have also been used without consideration for the change in weight and/or height seen with increasing age (Abraham and Campbell, 1980). Another way of presenting data is through the use of the percentile distribution of weight by age, sex and height (1981 Canada Fitness Survey, in Jetté, 1983).

1.2.2 Skinfold Measurements

Skinfold measurements provide a measure of subcutaneous fat in the trunk or abdominal regions depending on the various sites chosen. Ratios or sums using different skinfold measures have been used in the assessment of body composition for individuals or in population studies. Frisancho (1984) among others, has stressed the fact

that any definition of either obesity, normal weight or undernutrition should take into account the expression of body composition or the pattern of fat distribution. Android (male-like) obesity where fat is mostly deposited over the trunk and abdomen has been more strongly associated with increased morbidity, e.g. glucose intolerance, hyperinsulinemia, lipid disorders and mortality, than gynoid obesity (female-like) in which the predominance of fat is around the hips and lower limbs (Bray, 1980; Björntorp, 1985; Roncari, 1986).

A variety of different sums and ratios has been applied e.g. percentiles with triceps and subscapular skinfolds (Frisancho, 1984); sum of five skinfolds (triceps, biceps, subscapular, iliac crest and medial calf) (1981 Canada Fitness Survey); sum of three skinfolds, different for men and women, (Jackson and Pollock, 1985); subscapular/triceps skinfold ratio, (Foster et al., 1985).

Questions have been raised as to the advantage of applying such measures to the general public. For example, White and Periera (1985) have indicated that "in view of the wide variety of obesity types and inherent observer variation in applying this technique, the role of skinfolds in assessing obesity poses some theoretical and practical problems." Womersley and Durnin (1977) observed that skinfolds are more closely related to body density than height-weight indices for men and younger women but not for women in the age range of 30 to 49 years. The addition of other variables such as height-weight indices and limb circumferences greatly improved the accuracy of predictions of body density (White and Periera, 1985). However, both measures (skinfolds and height-weight indices) demonstrate epidemiological validity.

One could say that height, weight and weight/height^2 measurements are more precise than skinfold measures (Periera and White, 1985) which are in turn more precise than circumference measures. The position of the caliper and the pressure of the fingers on the skinfold while the measure is being taken are all factors affecting the precision of this measure (Léger, 1981).

Skinfold measurements are problematic in meeting the criteria for reliability. To increase reliability, skinfold measurements should be taken at several sites according to Womersley and Durnin (1977). The specific locale of a skinfold measurement should always be the same for consistent results.

Skinfold measurements are subject to a requirement for a quality calibration of the caliper which may be subject to metal fatigue if spring-loaded. Because spring-loaded skinfold calipers are more subject to decalibration and are more difficult to recalibrate properly, especially for the non-specialist, this measurement is not totally accurate. Specific training and skill are required in the use of skinfold calipers to ensure reliability and accuracy. Skinfold calipers can be very costly, and are not readily available in most Canadian households. The individual being assessed is often requested to wear certain types of clothing or to undress prior to measurement; consequently these measurements are not easily accepted by all individuals.

Despite potential difficulties in applying skinfold measures to weight assessments, skilled

professionals should continue to use this method to extend results generated using the BMI.

1.2.3 Circumference Other Than Waist-Hip

A useful review of various skinfold, circumference and weight-height indices has been prepared by Womersley and Durnin in 1977. As stated in Dr. White's report (1985), "these investigators compared these measurements with density and found in younger men and women that the use of several variables rather than skinfold thickness alone brings about a significant increase in the accuracy of predictions of body density." This type of measurement meets the following criteria: skill or simplicity of measurement, cost and availability as measuring tapes are available in most Canadian households. However, few population studies used circumferences other than waist and hip circumferences to study mortality and/or morbidity in a specific population. Therefore, this type of measurement cannot be considered at this time for development of new weight guidelines related to morbidity and/or mortality data for both men and women.

1.2.4 Waist-Hip Ratio

Measuring the ratio of the waist circumference in relation to the hip circumference provides a valid representation of the pattern of fat distribution. Abdominal obesity could be diagnosed by this more simple means. Abdominal or android obesity has been demonstrated consistently to be "strongly associated with risk factors for cardiovascular disease in cross-sectional investigations of older and more recent dates" (Björntorp, 1985). Skinfold measurements (sums or ratios) provide a direct measure of fat distribution but present numerous associated problems as described previously. "The greater reliability with which circumferences can be measured as compared to skinfolds would suggest that formulas using circumferences or diameters would be more reliable." (Bray, 1980).

Furthermore, recent epidemiological data have shown the possible use of the Waist-Hip Ratio (WHR) in the development of risk estimates regarding cardiovascular diseases (Björntorp, 1985; Blair et al., 1984), diabetes (Hartz et al., 1983) and hypertension (White et al., 1986a).

"More specifically, an increased waist-hip circumference ratio or waist-hip girth ratio (WHR) bears this more common correlation...the effects of regional distribution of adipose tissue may be independent of, and additive to, those of degree of obesity." (Roncari, 1986). Roncari adds that "... the waist-hip circumference ratio may be a better predictor of cardiovascular complications and related deaths than other measures of adiposity."

Actually, the WHR can be used to subdivide obese individuals into subgroups which may give a better estimate of their risk of developing some health problems. This is in agreement with the findings of White et al. (1986b) who found the Waist-Hip Ratio to be a "strong independent predictor of hypertension in both men and women" even though it only adds to the best prediction obtained with the Body Mass Index. Hartz et al. (1984) also suggested that the assessment of the impact of obesity or excess fat on disease should include some measure of the predominant location of body fat together with that of total body mass.

However, these data are of recent origin and require further validation. For example, the value for the ratio at which risk of morbidity and mortality increases significantly for both men and women of various age groups remains to be determined. Therefore, this measure is a reliable and simple one, but at the present time, it is too early to suggest any exact values as the "ideal" Waist-Hip Ratio. However, it could be used in association with a weight measure to assess fat distribution and to differentiate among obesity subtypes.

1.2.5 Weight-Height Ratios

There are a number of Weight-Height Ratio measurements such as the Ponderal Index, Wt/Ht and Wt/Ht^2 which have been applied to individuals and in population studies. Each measurement has strengths and weaknesses in assessing weight status.

White and Periera (1985) stated that "indicators based on a ratio of weight and height should provide a continuous scale of measurement that could be applied to any population or individual".

Khosla and Lowe (1967) evaluated three weight/height ratios: Wt/Ht, Wt/Ht^2, Wt/Ht^3. In the review by White and Periera (1985), they indicated that "the authors proposed that any index of obesity derived from measurements of weight and height must be: (1) highly correlated with weight, (2) independent of height. Wt/Ht^2 (Body Mass Index) was found to satisfy both criteria".

For example, the Ponderal Index (height divided by the cubic root of weight) used in the Nutrition Canada Survey (Health and Welfare Canada, 1973) was too strongly correlated with height. Consequently, there was an increase in the number of false positives, e.g. obese that are not obese, in a given population.

In most populations, weight increases directly with the height squared. The most precise method is to use a power function of 2 for men and 1.5 for women. However, White et al. (1986a,b) have analyzed blood pressure data of the 1981 Canada Fitness Survey (Canada Fitness Survey, 1981) using both formulae for women, i.e. $weight/height^2$ and $weight/height^{1.5}$. Their conclusion was that "although the new choice of categories regroups some of the women, there is no important difference in the apparent relationship between body mass and (mild) hypertension. Similarly, the changes in the correlation coefficients and the relative risk estimates are relatively minor, and our overall interpretation remains the same." Therefore, because the interpretation of results implies only minor changes when a power function of 2 instead of 1.5 is used and also, for the simplicity of the calculations, a power function of 2 for both men and women has been used in population studies.

Norgan and Ferro-Luzzi (in Simopoulos, 1985) suggest that there is a relatively high correlation between the Body Mass Index and body fat (as estimated from body density), particularly when age is taken into consideration. Therefore, in the absence of a direct measure of body fat, it is the most satisfactory measure based on weight and height that is available.

In reference to the criteria described in 1.1, BMI has been found "to have greater content validity than skinfold thickness but similar

concurrent validity. Even if at this time there is more evidence of predictive validity in terms of morbidity and mortality for weight indicators than for measurements of subcutaneous fat, further analysis of such data in relation to both measurements is needed" (White and Periera, 1985). Therefore, one could say that both the BMI and skinfold measurements have the same epidemiological validity. BMI presents a higher degree of precision, reliability, accuracy and client acceptability, i.e. easy and less time-consuming. BMI does not require special skills and the equipment (scales, measuring tape) is found in most Canadian households so that this measure is not costly. White and Periera (1985) have provided a detailed discussion on the BMI in their background paper: "Measurement of obesity and overweight in practical settings: an epidemiological perspective."

The BMI is a valid measure of weight in relation to health. It is not recommended for use in this report as the sole measure of either body composition or level of fitness.

1.2.6 Conclusion

Five measurements were assessed according to the criteria set by the Expert Group. The results of the assessment are presented in Table 1.

Body Mass Index is the only measure that meets all of the eight criteria selected as a basis for the choice of the best measurement to use in the development of new guidelines relating weight to health. However, it should be recognized that the BMI, on its own, has limitations.

Because of the increasing importance of the measurement of fat distribution in the prediction of risk of mortality and morbidity, the suggestion to include such a measure is sound. The measurement of the waist circumference divided by the hip circumference (Waist-Hip Ratio) has been put forward as a simple and relatively accurate measure. However, this measure has not yet been applied to many population studies and requires further investigation to assess its epidemiological validity. Until more precise values based on work such as that of Björntorp or a scale of measurements can be given, this measure can be used by health professionals as an indicator of fat distribution.

1.3 Application of Data Banks to the Selection of Weight Guidelines

In the development of new weight guidelines for the Canadian adult population, data banks have been considered from the perspective of the availability and validity of the data relating weight to major diseases (cardiovascular, cancer, diabetes) and/or risk factors (hypertension, high blood cholesterol and triglycerides) and/or mortality. For the purpose of this discussion, only studies using the BMI and/or WHR will be considered. A brief review of major prospective and retrospective studies from other countries will be followed by an analysis of the Canadian data banks.

For a more extensive review of the Canadian surveys and international literature, the reader is referred to the report by Stockwell and White (1984) on the "Application of Existing Canadian Data Bases to the Epidemiological Study of Under/overweight in Canada, in Reference to the Development of a Health Promotion Strategy", as well as to Appendices I and II of this report.

1.3.1 Review of Selected Prospective and Retrospective Studies Using the BMI and the WHR

A summary of the source, time of the survey, mortality and morbidity data collected, weight measurement used, description (number, sex, age) of the population surveyed and major findings and specifications of the major prospective and retrospective studies now available is given in Table 2 (mortality data) and Table 3 (morbidity data). More precisely, for each study reported in this section, the BMI and/or WHR related to the highest or lowest risk of morbidity and mortality are given when available. When specific BMI and/or WHR ratio values were lacking, trends suggested by the authors are described.

Few studies have used the BMI in relation to mortality. The duration of follow-up in these studies ranged from 10 to 26 years and most studies adjusted the data for confounders such as age, cigarette smoking and hypertension. BMIs associated with the lowest mortality are given in this section.

Morbidity is defined in Table 3 as the presence of diseases such as diabetes (Type II), cancer (breast, stomach or colon), arteriosclerosis, arthrosis, gout, varicose veins, digestive diseases, coronary heart disease and stroke, or of risk factors such as hyper-cholesterolemia, low HDL-cholesterol and high LDL-cholesterol concentrations, hypertriglyceridemia and hypertension. The duration of follow-up in these studies ranged from 6 to 26 years.

Most often, increased risk of morbidity is defined at higher levels of BMI. While few studies have considered the lower end of a BMI scale, a lower level of BMI is often related to confounding factors such as smoking habits. Studies in several countries show that the risk of premenopausal breast cancer appears to be inversely related to the BMI (see Table 3: Medalie et al., 1975; Paffenbarger et al., 1980; Lubin et al., 1985). However, the full relationship of a lower BMI to health has yet to be clearly described. This issue will be covered in section 2.1.

As can be seen in Tables 2 and 3, few studies have used the BMI in conjunction with a method for assessing fat distribution (WHR or skinfold measures). For the purpose of this review, studies using only skinfold measures have been deleted since this method was found to be inappropriate as a weight measure for the target population (see chapter IV, section 1.2). Most studies have used the BMI alone. In the studies using the WHR, few values are available. More research is needed before WHR values can be used by the general public and health professionals as the only values related to an elevated risk of morbidity and mortality. The principle of measuring fat distribution remains an important one and it will be discussed in section 2.2.

Finally, weight guidelines should ideally be developed from data of surveys with a sample similar or identical to the population of future users. Therefore, Canadian data banks are the most appropriate for the development of new weight guidelines for the adult Canadian population. Surveys of other populations are helpful in providing information needed for the final selection of specific cut-off points, as will be discussed in section 2.

1.3.2 Canadian Data Bases

Large data bases are needed for the development of weight guidelines linking some anthropometric measurement to morbidity and mortality data. Actually, recent prospective and retrospective studies have shown a relationship between some levels of BMI and/or WHR and morbidity expressed as the presence of risk factors and/or specific diseases. However, developing weight guidelines for a certain population is maximized by the use of data coming from the same population when available.

For a more complete discussion on the application of the Canadian data bases in the development of new weight guidelines, the reader is referred to the report by Stockwell and White (1984). Three major Canadian Surveys can be examined: The 1970-72 Nutrition Canada Survey (Health and Welfare Canada, 1973); the 1978-79 Canada Health Survey (Health and Welfare Canada, 1978) and the 1981 Canada Fitness Survey (Canada Fitness Survey, 1981). Another survey, the 1985 Health Promotion Survey (Health and Welfare Canada, 1985) is excluded because weights and heights were self-reported and no data on morbidity and mortality were available. Similarly, in a Manitoba study by Rabkin et al. (1977), men who were judged fit for pilot training in World War II were the reference population studied. This select reference population cannot be used at this point since it is not representative of the general Canadian population.

A. Nutrition Canada Survey (1970-72)

The Nutrition Canada Survey was conducted to assess the nutritional status of Canadians of all ages. Of the population studied, 7037 individuals were aged 20 years or more. The detection of abnormalities related to current or past malnutrition was the purpose of the clinical examination which included weight, height, biceps and subscapular skinfold measurements. There are definite limitations to using Nutrition Canada data in developing weight guidelines. In this study, morbidity data included: hypertension, but diastolic values were recorded only at a level of 100 mm Hg or greater; hyperlipidemia, although triglyceride data were of no value since the subjects were not in the fasting state; other signs specific to malnutrition; but general health was not assessed. The Nutrition Canada Survey had the lowest response rate (42.3%) for the adult population and this has been attributed to the fact that data collection was done outside the home in contrast to the other two surveys. Therefore, there is a higher risk of response bias, i.e. individuals with chronic diseases or with known malnutrition problems may have refused to undergo the assessment. This is a major reason why the Canada Fitness Survey (for hypertension) and the Canada Health Survey (for other illnesses or risk factors) have been retained for further discussion in the development of new weight guidelines.

B. Canada Health Survey (1978-1979)

The Canada Health Survey was conducted among non-institutionalized Canadians and included 10 571 households and represented an 86% response rate. An interview-administered and a self-administered questionnaire were used to examine current health status and risk factors to health. A subgroup representing one third of the selected population was further

investigated for physical measurements and serum analysis. This subgroup can be considered since height, weight and skinfolds, as well as blood pressure and cardiovascular fitness, were measured only in this subset population. The sample population falls to 4829 individuals. For individuals 15 years of age and over, the overall response rate was 60% for anthropometric data and 51% for serum samples.

C. Canada Fitness Survey (1981)

As suggested by its name, this survey was designed to describe the activity patterns and levels of fitness of the Canadian population. The survey included a self-administered questionnaire and anthropometric measurements (height, weight, skinfolds, girth and bone diameters). Data for anthropometric measurements and standardized tests of fitness were collected by trained technicians. The estimated response rate for the population aged 7 to 69 years in this survey is 58% (including 15 723 men and women from 20 to 69 years of age). Blood pressure was measured and therefore, morbidity data are available for hypertension only. Other data on diabetes, coronary heart disease, etc. were reported in the self-administered questionnaire. Some confounders such as cigarette smoking, alcohol consumption and sleep patterns have been considered in this survey. The quality of the anthropometric data and the large numbers of data collected make this survey an excellent data base*.

1.3.3 Application of Canadian Data Bases to the Selection of Weight Guidelines

Three Canadian surveys have been considered. Both the 1981 Canada Fitness Survey (CFS) and the 1978 Canada Health Survey (CHS) were used for further analysis concerning the epidemiology of over/underweight in Canada, i.e. prevalence and relationship of weight to health among Canadians. Both surveys provide cross sectional data, present some response biases (low response rate) and have a number of limitations described previously. The CHS data were used with some reservations and after some adjustments (Appendix I) to examine the association between levels of BMI and diabetes and other health indices while the CFS data were used for hypertension and BMI. Dr. White summarized the principal advantages and disadvantages of alternative approaches (using Canadian data or extrapolation from US studies) to population risk assessment (White, 1985). The advantages of using Canadian data are: external validity; a high degree of feasibility; reduced cost; possibility of validating the data.

Therefore, these data bases (CHS and CFS) are used for the subsequent discussion on the choice of cut-off points; the other prospective and retrospective studies are used to confirm the final decision. Since no Canadian data are available at the moment on the relationship between the WHR and morbidity data, the discussion will consist of a review

*Note: Diastolic blood pressures were taken at the fourth phase in the Canada Fitness Survey. Therefore, any analysis based on CFS diastolic blood pressure data may overstate the prevalence of hypertension in comparison with other surveys which used fifth phase data.

of international literature. However, a recent article from White et al. (1986a) has been included in the discussion on the use of the WHR as a predictor of hypertension.

1.3.4 Conclusion

As a background to the discussion on the rationale behind the choice of specific cut-off points for the BMI continuum, it is important to present the following conclusions:

1. The selected criteria for the choice of the "best measure" of weight in relation to morbidity and mortality data are the following: epidemiological validity, precision, reliability, accuracy, skill, availability, cost and client acceptability.

2. The BMI is selected as the primary weight measurement since it meets all of these criteria.

3. Fat distribution may be a better indicator of the presence of risk factors (hypertension, hyperlipidemia) or a better predictor of some diseases (coronary heart disease, diabetes, cancer) than the relationship of weight to height. Measures of fat distribution should therefore be considered in the development of these new guidelines.

4. Because the WHR meets most of the criteria previously described, it has been suggested to add this measurement as an indicator of fat distribution for use by health professionals.

5. Both the Canada Health Survey and the Canada Fitness Survey will be used in the selection of appropriate cut-off points on the BMI scale.

6. Major prospective and retrospective studies will be included in the overall discussion for both the BMI and WHR.

2.0 Selection and Interpretation of Cut-off Points

This section deals with the rationale behind the choice of cut-off points on the BMI scale and for the WHR values. The selection for the BMI scale has been made in response to the question "What levels of BMI are related to an increased evidence of mortality or of a specific health problem (e.g. hypertension, hyperlipidemia, coronary heart disease, diabetes, cancer)?" Tables 2, 3 and 4 should be consulted for an overall presentation of supporting information.

In the second part of this section, additional information related to the measurement of the Waist-Hip Ratio is given. It is important at this point, to refer the reader to the comprehensive report by Dr. Roncari (Appendix III) for a more complete understanding of the clinical-metabolic evidence for the impact of obesity on cardiovascular risk. While Dr. Roncari indicates that "At present, it is not possible to provide quantitative estimates of the impact of obesity on cardiovascular risk factors", he also summarizes his report by stating that "Persons with truncal obesity who are not usually massively corpulent have the strongest association with cardiovascular risk factors." Thus reviewing potential cut-offs for both the Waist-Hip Ratio and the Body Mass Index is essential.

2.1 Body Mass Index (BMI)

The Body Mass Index continuum was developed from the recognition that J-shaped or U-shaped curves exist which in addition to being age-related, relate morbidity and mortality to weight. J-shaped or U-shaped curves could be described as being curvilinear relationships, with increased morbidity or mortality present at one (J-shape) or both (U-shape) extremes (e.g. highest and lowest levels) of

the weight spectrum. In other words, if the lowest incidence of hypertension is seen in male adults 25 years of age with a BMI of 22, the same incidence does not hold for male adults aged 55 years and more. Tables 2 and 3 give results by age and sex. One can see that for a specific health problem, results vary according to age (trends/decades) and frequently by sex. Males are often more at risk than females of the same age and BMI values. This has to be kept in mind since the selection of BMI cut-offs could not be made for all factors such as age groupings/sex/risk factor/disease/mortality for very practical and obvious reasons. Therefore, the rationale behind the choice of cut-off points was based on epidemiological, clinical and metabolic evidence relating some BMI levels with the highest risk for all age groups and both sexes of developing risk factors of hypertension, hyperlipidemia and diseases such as coronary heart disease, diabetes and cancer.

2.1.1 Zone D: <u>Increasing Risk of Developing Health Problems (BMI greater than 27)</u>

The following section presents a specific review of four risk factors or disease conditions:

A. Hypertension

In the 1981 Canada Fitness Survey (Canada Fitness Survey, 1981), the prevalence of diastolic and systolic hypertension increases in males with a BMI greater than 27, particularly diastolic hypertension for those males aged 20 to 29 years and 50 to 59 years. There is an increase in systolic hypertension among women with a BMI greater than 27 at 30 to 69 years of age. The next major increase in the prevalence of both diastolic and systolic hypertension is seen at a BMI greater than 29 and greater than 31 depending on the age group and sex.

This trend is in agreement with Van Itallie's (1985) analysis of HANES II data showing that the risk of hypertension is 5.6 times higher in males with a BMI equal to or greater than 27.8 and in females with a BMI equal to or greater than 27.3 among those 20 to 45 years of age. The risk of hypertension is twice as high at the same BMI levels in those 40 to 75 years of age. Barrett-Connor and Khaw (1985) after a nine-year follow-up of hypertensive individuals, suggest that the incidence of mortality is higher in hypertensive men with a BMI below 27 than in men with a BMI greater than 27. The underlying explanation would be that a different mechanism is involved in the etiology of hypertension. In the obese patient "the increased stroke volume that augments cardiac output....is usually associated with an expansion of total blood volume. Elevated preload gives rise to left ventricular dilation and increasing ventricular volume. The compensatory increase in myocardial mass remains proportional to chamber dilation. As a consequence, at a given level of arterial pressure, total peripheral resistance is lower in an obese patient than in a lean one." (Barrett-Connor and Khaw, 1985).

B. Diabetes

In the 1978 Canada Health Survey (Health and Welfare Canada, 1978), the prevalence of diabetes increased with a BMI of 27 to 30 in women, with a second major increase in

prevalence observed at a BMI equal to or greater than 31. In men, the increase is seen only at a BMI equal to or greater than 31. Medalie et al. (1975) confirmed the higher incidence of diabetes in 50- to 59-year-old men with a BMI greater than 27. In the analysis of HANES data, Van Itallie (1985) found the risk of diabetes was 3.8 times higher for those 20 to 45 years of age and about twice as high for those 45 to 75 years of age when the BMI was equal to or greater than 27.8 for men and 27.3 for women.

C. Cholesterol

Analysis of the 1978 Canada Health Survey data indicates that after adjusting for age, the prevalence of hypercholesterolemia is higher in women than in men with a BMI greater than 27. It is, however, higher in men than in women with a BMI less than 25. Simpson et al. (1982) found a sharp increase in serum cholesterol levels only in men aged 20 to 49 years with a BMI greater or equal to 28.5; the increase in serum cholesterol levels is gradual for women. Farinaro et al. (1979) found the highest levels of VLDL-triglyceride and LDL-cholesterol in men with a BMI greater than or equal to 28. In general, cardiovascular problems are associated with increasing levels of BMI when there has been a long history of obesity, e.g. 16 years in the Manitoba study (Rabkin et al., 1977), and 6 years in the Framingham Study (Sorlie et al., 1980) and when the population is a younger age group of men.

D. Cancer

The relationship of body weight to cancer depends on the type of cancer and the person's age. Risk of breast cancer with increasing BMI increases in post-menopausal women and decreases in pre-menopausal women (Table 3). A reversed J-shaped curve was observed in the Japan-Hawaii Cancer Study for stomach cancer in males in age groups younger than 55 years and older than 55 years with BMIs less than 24.6 and greater than 26.3 (Nomura et al., 1985).

In conclusion, these studies show that BMIs greater than 27 are associated with an increased occurrence of hypertension, hyperlipidemia and coronary heart disease. A second increase is observed with a BMI greater than 30. For this reason, a BMI greater than 27 has been suggested as associated with an increasing risk of developing health problems for both men and women.

The response to a higher BMI varies according to the various age groups but with the present state of knowledge, it is impossible to set cut-off points for specific age groups. However, there is a trend for a stronger impact of obesity in the younger age group if obesity is present over a period of many years. The CFS data suggest that, for men, BMI is a stronger predictor of hypertension than age (Table 3).

2.1.2 Zone C: May Lead to Health Problems in Some People (BMI between 25 and 27)

Contrary to what is observed in Zone D, no sharp increase in health problems is associated with a BMI of less than 27. No Canadian data support the choice of cut-off points below 27 (Table 3). However, the risk of developing stomach cancer increases at a BMI greater than 26.3 in the Japan-Hawaii Cancer Study (Nomura et al., 1985) and at a BMI greater than 25 for digestive diseases (Bray, 1985).

While no specific cut-off points are given, many authors of the studies in Table 3 suggest an increasing risk of developing some health problems with increasing levels of BMI. Since sharp increases in morbidity and mortality have been reported for a BMI greater than 27, it seems logical to suggest a caution zone as the BMI increases to 27. Bray (1985) suggests that low-risk zones should be at a BMI between 25 and 30 with the lowest risk zone being at BMIs between 20 and 25.

In conclusion, since sharp increases have been reported for some morbidity/mortality data with BMIs around 27, the "caution" zone should cover ranges of BMIs from 25 to 27. This will be described as the zone where "overfatness" may lead to health problems in some people.

2.1.3 Zone B: Good Weight for Most People (BMI between 20 and 25)

Waaler (1984) showed that a BMI of 23 to 25 for men and one of 23 to 27 for women was associated with the lowest mortality rate in his study population of 20 years and over (Table 2). For men aged 30 to 49 years, the highest mortality rate was found at BMIs equal to or greater than 31. In the Seven Countries Study, Keys (1980) showed the highest mortality rates for men 40 to 59 years of age at BMIs equal to or less than 22 and equal to or greater than 32. Jarrett et al.'s (1982) analysis of the Whitehall Study, demonstrated that the highest mortality in men 50 to 59 years of age was at BMIs equal to or less than 22 and greater than 27.

However, when confounders are taken into account, other studies do not show the same trend for cut-off points of a BMI around 22. Despite the various J-shaped or U-shaped curves associated with age, there is agreement that when both men and women are considered, the highest mortality reported was associated with BMIs of less than 19 and greater than 31 but most often greater than 27. Based on mortality data from various population studies, Andrés et al. (1985) suggests that "a range of 20 to 25 kg/m^2 on the index is now frequently recommended as a standard of normality for body weight". This recommendation is in agreement with Bray's lowest risk zone of 20 to 25 (Bray, 1985).

A decision is further complicated by the fact that morbidity curves demonstrate a different pattern than mortality curves; some curves show an increasing risk at a BMI of less than 21 or even at 22 or 23 (Appendix II).

The risk of developing cancer is the most difficult to evaluate for the BMI category of 20 to 25. For some cancer states, e.g. breast cancer in pre-menopausal women, the risk decreases with increasing BMIs. However, breast cancer in post-menopausal women increases with increasing BMIs.

In conclusion, a range of BMIs between 20 and 25 for Zone B takes into consideration variations according to age group/sex/morbidity data/mortality data. It is also in agreement with the most recent literature on the standard of normality for body weight. Consequently the range of 20 to 25 represents a good weight for most people.

2.1.4 Zone A: May Be Associated with Health Problems for Some People (BMI less than 20)

This zone, which includes BMIs of less than 20, is not representative of the same morbidity risks as the higher BMI levels. The causes for mortality associated with a BMI of less than 20 are also different. In Table 2 one can see that the lowest mortality for both men and women is most often associated with a BMI of less than 20, sometimes even 22 and 23. This trend is not age-dependent. Furthermore, underlying pathologies such as cancer could be the cause of this lower BMI and, as a result, this zone of BMI is difficult to interpret. In other words, a decreasing BMI may be the result of the disease rather than the cause of the disease.

In the Health Promotion Survey (Health and Welfare Canada, 1985) over 50% of Canadians wanted to change their weight, primarily through weight loss. About 70% of women at a "good weight" (BMI 20 to 25) wanted to lose weight as did 23% of women with a BMI less than 20.

Since there is an increase in the prevalence of eating disorders such as anorexia nervosa and bulimia in both adolescent and adult groups, particularly for younger women and men, the "entry" into Zone A could be used as a trigger to search for the underlying cause of a lower BMI. Rapid movements down the scale of BMI to Zone A should be perceived as the time to investigate reasons behind this rapid change in body weight.

However, it is necessary to note that some athletes or professional dancers, and some women with very small frames may have a BMI lower than 20 and may maintain this level of BMI for decades. This cannot be associated with the health problems described previously and interpretation of BMI values with these individuals should be made with caution.

2.1.5 Canadian Guidelines for Healthy Weights

On the next page is a figure of the Body Mass Index continuum. Indicated on the figure are the four BMI zones for healthy Canadians 20 to 65 years of age.

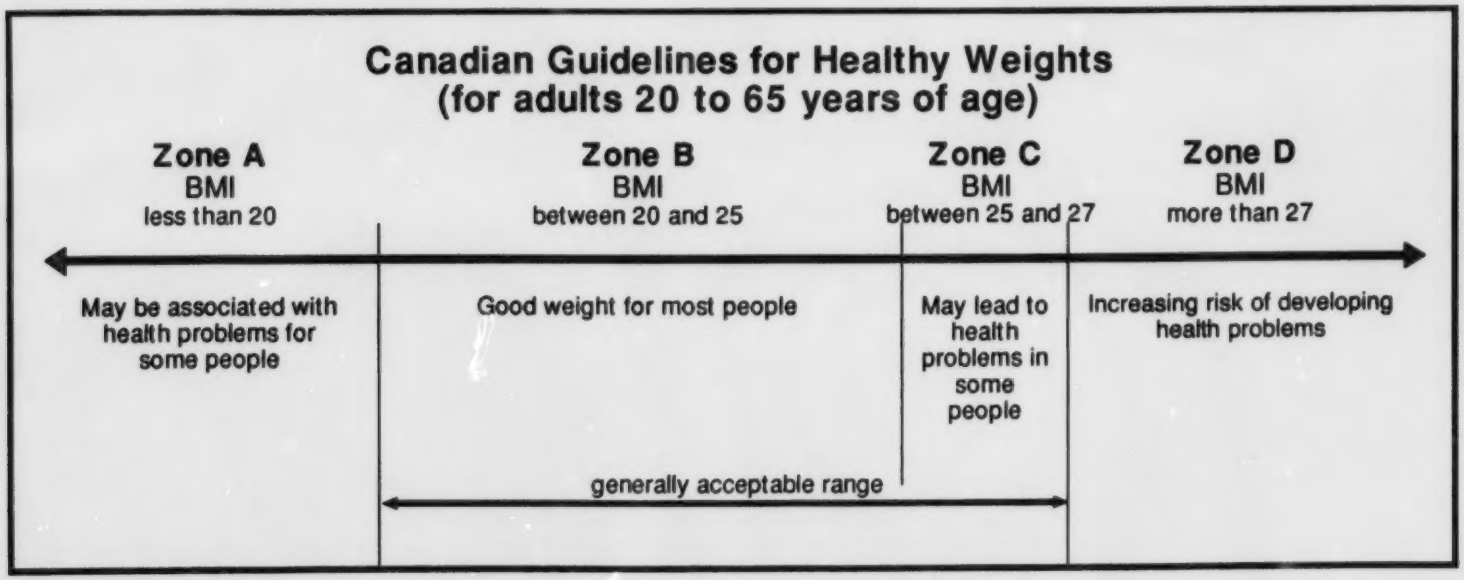
2.1.6 Interpretation Considerations

1. The Body Mass Index (BMI) depends on measures of weight and height. Both of these measures are easily accessible and highly accurate.

2. The BMI scale should be used as a "continuum" where the risk of developing health problems increases with shifts away from the "generally acceptable range".

3. Rapid changes in weight within and between zones should be considered as important indicators of potential problems particularly at the outer ends of the scale.

Figure 1


Canadian Guidelines for Healthy Weights
(for adults 20 to 65 years of age)
Zone A
BMI
less than 20
Zone B
BMI
between 20 and 25
Zone C
BMI
between 25 and 27
Zone D
BMI
more than 27
May be associated with health problems for some people
Good weight for most people
May lead to health problems in some people
Increasing risk of developing health problems
generally acceptable range

4. The BMI has been used primarily to identify high-risk groups in a specific population. Interpretation of the BMI for factors such as age and duration of weight problems is essential in applying this measure to individuals.

5. Because of variations in the association of BMI to risk factors and/or specific diseases, it is important to consider the fact that the choice of cut-off points represents the best choice to date based on data available at the present time. Young males with a BMI of 27 and over should be particularly concerned about an increased probability of developing health problems.

6. The same cut-off points for the Body Mass Index, $Wt(kg)/Ht^2(m)$, are appropriate for both men and women 20 to 65 years of age.

7. In the Canadian Guidelines for Healthy Weights, BMI is being used to reflect weight in relation to health. However, the BMI alone is not a direct measure of body fatness. It should be recognized that a BMI above the "generally acceptable range" of weight for health is not necessarily indicative of excess fat. A short stocky muscular individual, for example, may have a BMI above 27 yet not be at high risk for his/her health. At the same time, a BMI below the "generally acceptable range" of weight for health is not necessarily indicative of insufficient body fat and increased risk. For example, health endurance athletes may maintain a BMI below the "generally acceptable range" for years. Interpretation of BMI values in these cases should be made with caution.

2.2 Waist-Hip Ratio

Measures of fat distribution are often needed to confirm the impact of "overfatness" on cardiovascular disease, cancer, diabetes or on risk factors such as hypertension and hyperlipidemia. In cases such as glucose intolerance or high triglyceride levels, indices of fat distribution are better predictors than weight measures such as the BMI, Relative Weight or Broca Index alone. For example, "certain cardiovascular risk factors are more commonly associated with obesity characterized by disproportionate accumulation of adipose tissue in a "truncal" or "abdominal" distribution (Appendix III). The metabolic consequences of particular adipose tissue distributions are well described in Appendix III and might be related to various causes such as regional specialization of fat deposits on growth and differentiation of adipocyte precursors, on the size of mature fat cells and on adrenergic receptors and variations between "region" in the expression of metabolic properties.

In the Gothenburg study (Larsson et al., 1984; Lapidus et al., 1984), the results for the WHR were the anthropometric measurements which differentiated between cases and non-cases rather than BMI and skinfolds. In men, a change from the lowest WHR ratio (Quintile I) to the highest ratio (Quintile V) corresponding to a BMI of 22.5 to 27.7 was associated with an increase in systolic and diastolic blood pressure, and in serum cholesterol levels. In women, a change from Quintile I to Quintile V (corresponding to a BMI of 22.5 to 27.0) was associated with an increase in systolic blood pressure, total serum cholesterol and blood glucose (White and Periera, 1985). Roncari, (1986) (Appendix III) has indicated that "Persons with truncal obesity who are not usually

massively corpulent, have the strongest association with cardiovascular risk factors." The same trend was reported by Björntorp (1985) who suggests that when the WHR exceeds 1.0 in men and 0.8 in women, the risk for cardiovascular disease seems to increase steeply.

High BMIs are not always related to "overfatness". Probably one could say that all obese people are overweight, but not all overweight people are obese or "overfat".

An additional measure such as the WHR could determine the presence or absence of overfatness and the distribution of excess fat.

At this moment, few studies have included measurements of fat distribution in addition to measures of BMI or of Relative Weight to determine precisely the impact of fat distribution on morbidity and/or mortality. Roncari, (1986) (Appendix III) summarizes the relationship as "in general, progressively greater degrees and duration of truncal obesity have increasingly more pronounced association with cardiovascular risk factors. However, this positive relationship is by no means linear because a variety of genetic environmental factors interact to modify the expression and impact of the cardiovascular risk factors."
Björntorp (1985) suggested specific WHR values:

1.0 for men
0.8 for women.

There is increasing risk with increased values but at this time no scale of measurement or "continuum" showing increasing probability of developing health problems is available.

In conclusion, the values suggested by Björntorp (1985) could be used as indicative of an increased risk. However, much more research on this topic is necessary before the Expert Group can give more detailed recommendations on the use of the WHR.

2.3 Conclusion

The Body Mass Index (BMI) is a measure which is epidemiologically valid for public health purposes from the perspective of determining high-risk groups. The BMI could also be used by the general public as a measure that describes the chance of developing specific health problems considered in the development of these new weight guidelines. The addition of a measure of fat distribution (WHR) is often a better predictor than the BMI alone of the chance of developing these health problems, particularly cardiovascular disease. Therefore, it is recommended that four zones of the BMI be used for the weight-health standards and the WHR as an indicator of fat distribution, in view of its relationship to cardiovascular risk factors.

Note: Fitness Canada has adopted the BMI as one of four measures which together can be used to assess body composition in a procedure entitled "Body Weight, Adiposity and Fat Distribution." This procedure uses the BMI in conjunction with skinfold measures by skilled appraisers for more precise determinations of body fat and fat distribution. For more information, readers are referred to the Canadian Standardization Test of Fitness (Fitness Canada, 1986). Training workshops on the use of the Canadian Standardized Test of Fitness are also available through the Fitness Appraisal Certification and Accreditation program (FACA). Information on FACA is available through:

The Canadian Association of Sports Sciences FACA Program
333 River Road
Ottawa, Ontario
K1L 8H9

V. APPLICATION OF THE BMI SCALE TO ESTABLISH A HEALTHY WEIGHTS PROFILE OF CANADIANS

In Canada, there has been growing interest over the past decade in the prevalence of weight-health problems in terms of obesity, overweight and underweight.

The prevalence of these problems, applying the suggested BMI cut-off points to the Canada Fitness Survey (CFS) and Canada Health Survey (CHS) is presented in Table 5. The CFS (Canada Fitness Survey, 1981) and CHS (Health and Welfare Canada, 1978) data represent the most recent weight "profile" of the Canadian population.

The difference in prevalence rates between the two surveys within age and sex groups may generally reflect sampling variability. The fact that the two surveys were conducted at two different periods of time and used different sampling designs may also contribute to the observed differences in prevalence rates between both surveys.

According to the Canada Fitness Survey, approximately one out of two adult Canadians are at a "good weight" for health (Zone B). More men (29%) than women (19%) are at "increasing risk of developing health problems" (Zone D). More women (15%) than men (5%) are at the lower end of the BMI scale which puts them in a category which "may be associated with health problems for some people" (Zone A). Both men and women are at "increasing risk of developing health problems" (Zone D) with increasing age.

For young adult males, the clinical and metabolic impact of obesity is most important. For young females, underweight is often a consequence of either eating disorders (excessive dieting, anorexia, etc.) or smoking habits, or a combination of the two. Consequently, it is of public health interest to point out the importance of preventing weight problems and promoting healthy weights, especially among young adults.

VI. RECOMMENDATIONS FOR THE IMPLEMENTATION OF THE CANADIAN GUIDELINES FOR HEALTHY WEIGHTS

At their first meeting, the Expert Group on Weight Standards recommended that two distinct documents, one for the general public and one for health professionals, be developed and tested.

The weight guidelines could be used by the public for establishing realistic, practical and meaningful body weights or images of healthy body weight. They could also aid in understanding the relevance of weight control to health status. This would be particularly important for younger women with a distorted image of body weight and for younger men who have a higher risk of developing health problems when overweight.

Health professionals could use the weight guidelines for: assessment of the degree of over- or underweight of clients and its impact on their health status; development and implementation of subsequent counselling and education programs for those clients who suffer from weight problems which may present risks to their health.

1.0 Document for the Public

Discussion by the members of the Expert Group on the document for the public is summarized as follows:

1.0.1 Target Group

The general public, including motivated and informed leaders, volunteers and media.

1.0.2 Content Outline

Section 1 Why guidelines for healthy weights

Key points:

to encourage appropriate, realistic weights and body images as related to risk to health

to discourage patterns of low or excess weight related to height

to respond to increased preoccupation with weight and to exploitation of weight problems

to address normal weight fluctuations.

Section 2 Who are the guidelines for healthy weights for

Key Points:

healthy adults 20 to 65 years of age, <u>not</u> for children, adolescents, pregnant women or adults over the age of 65

Section 3 What are the guidelines for healthy weights

Key Points:

BMI as a measure of weight and as a predictor of health. It is a more appropriate measure than the past use of weight-height tables.

fat distribution - distribution of fat on the body is also important; accumulation of fat around the waistline is more likely to lead to health problems

risk factors/health problems - e.g. increased cholesterol levels, diabetes, high blood pressure are predictive of heart disease and stroke and are associated with increasing fatness

obesity - excess body fat

underweight - insufficient body fat and its relationship to health.

Section 4 How to use guidelines for healthy weights

Key Points:

accurate measurements of weight and height are essential to calculate BMI

weight - it is preferable to weigh oneself on Canadian Standards Association (CSA) approved scales in the morning before breakfast. Frequent measurements are not necessary.

Note: There was considerable discussion as to whether to suggest a desirable frequency of weighing. If an accurate BMI was required, then the more frequent the measures, the greater the likelihood that an accurate BMI would be recorded. The use of "preferably not more than once per week" was considered by some to be associated with intervention rather than assessment. The issue of seasonal variation i.e. weight at four times per year also arose. In this section on "how to", the general statement "frequent measurements are not necessary" was acceptable.

height - take height without shoes, heels against wall.

Section 5 How to determine the BMI

Key Points:

The use of scale alone, nomogram alone, or a combined nomogram with scale was discussed. A nomogram (see sample nomogram in Figure 2) or other tabular or graphic presentations of the guidelines for healthy weights would be available for testing and inclusion in the final document for the public. Artistic rendering would be required as well as development of instructions.

Considerable discussion ensued as to whether to include the terms/-measures for obesity on the scale. For example, it was suggested that it would be meaningful to the public if a BMI greater than 27 was expressed related to obesity/fatness as well as to health. This is a complex issue for the public to understand at the present time. The emphasis on health and health promotion encourages the use of acceptable limits. Interpretation of BMIs less than 20 and over 27 will focus on the relationship of under- and overweight to health. The outcome of the discussion was to retain the scale as it stands.

Section 6 What do guidelines for healthy weights mean

Key Points:

If the BMI is between 20 and 27, the individual is in the "generally acceptable range" of weight for health. A BMI between 25 and 27 represents a caution area for some people because they are approaching increased risk to health with increased weight. Beyond a BMI of 27, most individuals are obese with progressive risks to health.

For some individuals, a BMI of less than 20 may be normal. On the other hand, a BMI less than 20 or a sudden decline in BMI may be associated with serious underlying health problems. Medical advice should be sought.

Rapid changes in BMI between different zones and also within each zone may signal health problems.

Further development of the following points is suggested:

additional points as qualifiers need to be developed

health impact of excessive weight should concern everyone but more so younger and middle-aged adults

frequency of BMI measurements for example, suggest twice a year to confirm weight for health

referral to the Report of the Task Force on the Treatment of Obesity for BMIs over 27

change in clothing, i.e. belt size, good indication of particular problem in fat distribution regardless of BMI

limitations to use of BMI scale e.g. athletes

health impact of low body weight

body weight partly reflects eating and physical activity habits; however, no two individuals are alike.

1.0.3 Implementation Strategies

Practical Suggestions for Presentation:

use of slide rule for public

use of wheel to consolidate information, e.g. Canadian Heart Foundation tool

cooperate with Metric Commission, i.e. use of their tapes for height measurements

commentaries should be included with scale

Actions Recommended by Expert Group:

field trial of the use of terms and understanding of BMIs; of nomogram, scale, or nomogram plus scale to test public acceptance and understanding.

2.0 Document for Health Professionals

The document for health professionals was not formally discussed at the second consensus meeting. However, suggestions had been given throughout the two consensus meetings and are summarized. It was recognized that the report of the Expert Group on Weight Standards would be distributed on a limited basis to a scientific health professional audience including researchers and academics involved in medicine, nutrition, nursing and physical education.

2.0.1 Target Group

Health professionals and policy makers.

2.0.2 Content Outline

The table of contents should be basically the same as the one proposed for the general public. However, each item should be more detailed.

The rationale section (chapter IV, section 1.0) for the choice of measurement and cut-off points should be included in total in the document made available to health professionals.

Values of the Waist-Hip Ratio (WHR) and items listed to help the interpretation of data are also important to include.

2.0.3 Implementation Strategies

The coordination and cooperation of provinces, health professionals and corporations will be necessary to implement these recommendations.

VII. RECOMMENDATIONS FOR FUTURE RESEARCH

Future research needs were not specifically discussed by the Expert Group on Weight Standards during their consensus meetings. However, suggestions for future research have been given throughout discussions at the meetings and in response to the draft report. In addition, recommendations for research will be brought forward by the Task Force on the Treatment of Obesity.

Research is needed:

1. to obtain more information on the morbidity/mortality profile of individuals at the lower end of the BMI scale (BMI less than 20)

2. to determine levels of the WHR at which the risk of morbidity/mortality increases significantly for both men and women of various age groups

3. to update data as it becomes available on the cut-off points and the rationale for cut-off points

4. to obtain reference values for the younger population below 20 years of age and older population over 65 years of age. The target group for these new weight standards is the healthy adult population 20 to 65 years of age

5. to determine the nature and mechanisms of genetic factors rendering subjects susceptible to the development of obesity

6. to determine how these genetic factors interact with environmental stimuli

7. to gain knowledge regarding inter-individual variability related to a) genetic factors and b) their interaction with environmental stimuli.

SUMMARY DEFINITION OF KEY TERMS

Risk Factor

"Attribute or exposure that is associated with an increased probability of a specified outcome, such as the occurrence of a disease...a "risk marker". It is also considered as an attribute or exposure that increases the probability of occurrence of diseases or other specified outcome...a determinant" (Last, 1983).

In disease prevention, this term includes social, biological or economic status, behaviour or other states known to be associated with or the cause of increased susceptibility to specific disease, ill health or accident. For example, in the context of this report, risk factors refer mainly to cardiovascular risks (hypertension, hyperlipidemia).

Health Problems/Disease Condition

They are problems or specific diseases such as hypertension, hyperlipidemia, coronary heart disease, diabetes, cancer.

Body Mass Index (BMI)

Is the expression of the weight (kg) divided by the height (m) squared (Wt/Ht^2: Weight/Height x Height). It incorporates weight from all body sources, including fat. The BMI has a high correlation with body fat (as estimated from body density) particularly when age is taken into consideration. In the absence of a direct measure of body fat, it is the most satisfactory index based on weight and height.

Fat Distribution

Location of body fat.

Waist-Hip Ratio (WHR)

Is an indicator of fat distribution. It is the waist circumference divided by the hip circumference. The Waist-Hip Ratio can be used to subdivide obese people into sub-groups which may give a better estimate of their risk of developing some health problems.

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Tables and Figures

TABLE 1
APPLICATION OF SELECTED CRITERIA IN THE ASSESSMENT OF WEIGHT MEASUREMENTS

Criteria / Measurement	Epidemiological Validity		Precision	Reliability	Accuracy	No Special Skills Required	Easily Available	Low Cost	Client Acceptability
	Mortality	Morbidity							
Weight-Height Tables	x	-	-	-	-	x	x	x	x
Skinfold Measurements	x	x	x	±	±	-	-	-	-
Circumference Other Than Waist-Hip	-	-	-	x	x	x	x	x	x
Waist-Hip Ratio	-	x*	±	±	x	x	x	x	x
Weight-Height Ratio:									
Body Mass Index	x	x	x	x	x	x	x	x	x

x = yes
- = no
± = more or less
* Based on recent studies

TABLE 2
DATA BANKS: Mortality Data

COHORT DESCRIPTION

Source	Time	Mortality	Measurement	N	Sex	Age	Major Findings	Specifications
Waaler (Norway)	1984	X	BMI	1 717 515	M, F	20 and over	· Lowest mortality M: BMI 23-25 F: BMI 23-27 · Increase in rate at BMI > 29 (F,M) or > 31 (M) depending on age group · M(30-49 years of age): Highest mortality with BMI ≤ 19 and ≥ 31	· No adjustments were made for cigarette smoking, hypertension · Follow-up 10 years in Norway
Jarrett et al. Whitehall Study (England)	1982	X	BMI	19 393	M	40-64	· Highest mortality BMI < 22 and > 27 for 50-69 years old	· Follow-up: 10 years. · Linear association BMI and mortality for those 40-49 years old Linear Negative: association between BMI and mortality for those 60-64 years old · Independent effect of weight on CHD mortality is negligible after adjustments for blood pressure, cholesterol, cigarette smoking

TABLE 2
DATA BANKS: Mortality Data (Continued)

COHORT DESCRIPTION

Source	Time	Mortality	Measurement	N	Sex	Age	Major Findings	Specifications
Keys Seven Countries Study (USA., Finland, Japan, Southern and Northern Europe) BMI	1980	X	BMI		M	40-59	· Negative relationship between BMI and all causes of mortality in Southern Europe and USA · Highest mortality with BMI ≤ 22 and ≥ 32 · Lowest mortality with BMI 28 to 32	· Samples from USA, Finland, Japan, Northern and Southern Europe . Follow-up 10 years · Relationship between weight and mortality was curvilinear (U.S. railroad men) after adjustment for age, systolic blood pressure, serum cholesterol, smoking, pulse rate
Sorlie et al. Framingham Study (USA)	1980	X	BMI		F M		· Lowest mortality with BMI 21 to 31 · Lowest mortality with BMI 32 to 38 · When taking confounders into account, no coherent pattern among males, and elevated mortality in females could be found with BMI < 21 and > 31	· Follow-up: 18 years · Negative relationship between relative weight and mortality for both M and F

TABLE 2
DATA BANKS: Mortality Data (Continued)

COHORT DESCRIPTION

Source	Time	Mortality	Measurement	N	Sex	Age	Major Findings	Specifications
Dyer et al. Chicago Peoples Gas Company (USA)	1975	X	BMI	1 237	M	40-59	· Lowest mortality with BMI 27 to 29	· Follow-up: 14 years Adjusted for age, systolic blood pressure, serum cholesterol, cigarette smoking; significant quadratic relationship was found between BMI and all causes of death (Cardiovascular; renal death; coronary heart disease)

TABLE 3
DATA BANKS: Morbidity Data

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Seidell et al. (Netherlands)	1986	Diabetes Arteriosclerosis disease Arthrosis Gout Varicose Veins	BMI (W/H^2)	444(M) 627(F)	M,F	20-50 years of age at the beginning of study	·For all diseases, except gout, risk increases with BMI > 30 · Risk of diabetes and arthrosis increases with BMI > 30 in M; of gout and arteriosclerotic disease with BMI 27-30 in M · Undiagnosed overweight subjects have relatively lower incidence of illnesses compared to diagnosed overweight subjects	· Follow-up 6 to 17 years · Inverse relationship between level of occupation and body fatness · Lower BMI considered was $\leq$ 27 · No adjustments were made for smoking, underweight or type of fat distribution
Barrett-Connor & Khaw (USA)	1985	Hypertension	BMI	1 727	M	50-79	· Men with BMI < 27 and hypertension have higher mortality rates than men with BMI $\geq$ 27 and hypertension	· 9 year mortality rates with diastolic blood pressure < 95 and $\geq$ 95

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Bray Build Study data (1979) (USA)	1985	Digestive disease and mortality	BMI				· "Digestive disease, primarily that of the gallbladder, ranked next to diabetes in degree of detrimental effect of excess weight" · J-shaped curve showing a decreasing effect from BMI 20-25 and a sharp increase with BMI $\geq$ 25 · J-shaped curve reflects the relation between BMI and the magnitude of the various complications reviewed in this article e.g. digestive diseases, pulmonary problems, endocrine disorders, obstetrics, orthopedic and dermal difficulties, renal functions and miscellaneous abnormalities. Bray suggests as a conclusion that for a BMI 20-25 lowest risk BMI < 20 and 25-30 low risk BMI 30-40 moderate risk BMI > 40 high risk	

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Foster et al. (USA)	1985	LDL-C HDL-C TG	Skinfolds · Thigh/Arm cir (U/L) · Subscapular/ Triceps (C/P) · Chest/Height Ratio	399	M, F	Adults	· High TG is mostly explained by the Upper/ Lower (U/L) fat pattern · High LDL-C is mostly explained by age · High HDL-C is mostly explained by a lean body mass · The central/peripheral fat (C/P) pattern comes second to explain the three measurements (TG, LDL-C and HDL-C)	
Lubin et al. (Israel)	1985	Breast Cancer	BMI	Cases 431 Controls 372	F	60 + years	· Risk increases with increase in BMI	· Results persist after adjustment for age at menarche, first birth, and menopause, years of education, number of births, history of benign breast disease, family history of breast cancer

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Nomura et al. Japan-Hawaii Cancer Study	1985	Stomach and Colon Cancer	BMI	8 006			·Positive relationship between colon cancer and BMI > 23.02 for M ≥ 55 years of age	· Follow-up 15 years · Prostate cancer had a weak association with BMI
							· Reversed J-Shape curve observed for stomach cancer in both age groups, risk increases with BMI < 24.6 and > 26.3	· Significant relationship between weight gain and colon cancer was observed only in men aged 55 · Weight gain was a better predictor than a high BMI throughout life
Van Itallie (USA)	1985	Hypertension Hypercholesterolemia Diabetes	BMI	NHANES II data			· Risk of hypertension is 5.6 times higher in overweight people 20 to 45 years of age · Is 2 times higher in overweight people 40-75 years of age · Risk of hypercholesterolemia: 2.1 times higher in overweight people in the 20-45 age groups · Risk of diabetes: 3.8 times higher for overweight people 20-45 years years of age; 2 times higher for overweight people 45-75 years of age · All risks remain higher even if skinfolds measurements are added to the BMI to describe fat versus lean mass	· Overweight if BMI ≥ 27.8 M ≥ 27.3 F Severely overweight if BMI ≥ 32.1 M BMI ≥ 32.3 F 2· Hypertension = systolic ≥ 160mm Hg and/or diastolic ≥ 95mm Hg · Hypercholesterolemia: Blood cholesterol ≥ 250mg/dl · Diabetes: diagnosed with a 2-hour glucose tolerance test

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Larsson et al. Gothenburg Study (Sweden)	1984	Stroke, CHD, Death	BMI WHR Skinfolds	855	M	Adults	· Compared to BMI and skinfolds, only WHR differed between cases and non-cases. Mean WHR was 0.97 ± 0.054 · Ratio of the highest and lowest quintiles was 2.5 for CHD and 5.9 for stroke · To obtain a positive trend for serum cholesterol and systolic blood pressure, BMI and skinfold had to be included in the analysis	· Follow-up 13 years
Hubert et al. Framingham Study	1983	Cardiovasc.	RW	2 252 2 818	M F	26-62 at entry	· Excess risks associated with relative weight for those under 50 years of age · After adjustments for other risk factors, an association was found for: M: CHD, angina pectoris, sudden death, congestive heart failure. F: CHD: angina pectoris, myocardial infarction, congestive heart failure and stroke	· Follow-up 26 years

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Krotkiewski et al. (Sweden)	1983	CHD	WHR	260 670	M F	Adults	· A higher WHR was a better predictor of CHD both in M and F	
LaVecchia (Italy)	1982	Breast Cancer	BMI	Cases 173 Controls 347	F	Premeno-pause	· Risk increases with BMI > 25	
Simpson et al. (Australia)	1982	Cholesterol	BMI		M, F	20-69	· Increase in cholesterol with BMI 28.5 in M aged 20-49 only · Increase in cholesterol more gradual in F	

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Fitness Canada, 1981 Canada Fitness Survey	1981	Hypertension	BMI (W/H^2) WHR	15 723	M, F	20-69	· Diastolic hypertension (> 90mm Hg) increases in M with BMI > 27 aged 20-29, 50-59 · A second major increase in M with BMI > 29 aged 30-49 and 60-69 · Systolic hypertension (> 140mm Hg) increases in M with BMI > 27 aged 20-29, and with BMI > 29-30 aged 40-49 · Diastolic hypertension increases in F with BMI > 31 aged 20-49 BMI > 29 aged 50-59 BMI > 27-28 aged 60-69* *may be due to medication. · Systolic hypertension increases in F with BMI > 31 with BMI 27-28 aged 30-69	· For men, BMI is a stronger predictor than age · For women, age is powerful · Greater effect of BMI in older age groups WHR contributor to diastolic hypertension · Age and BMI are more associated with systolic than diastolic hypertension

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Paffenbarger et al. (USA)	1980	Breast Cancer	BMI	1 098 premenop. 2 807 postmenop.	F		· Risk of breast cancer increases with increasing BMI in post-menopausal women (21.5< 24.4 < 24.5 and +); decreases with increasing BMI in pre-menopausal women (21.5> 24.4 > 24.5 and +); increases with amount of weight gain since age 20	
Farinaro et al. Olivetti Study (Italy)	1979	Plasma lipoproteins	BMI	107	M		Highest VLDL-triglycerides and LDL-cholesterol in men with ≥ 28	

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Health and Welfare Canada, 1978 Canada Health Survey	1978	Hypertension Diabetes Cholesterol Uric Acid SGOT Glucose	BMI (W/H^2)	3 622	M, F	20-69	· Results for hypertension do not follow a logical pattern e.g. in M: gradual increase up to BMI 25-26, drop with BMI 27-28, increase BMI > 29 +	· Those individuals (386) at both extremes of the BMI scales were eliminated for this analysis (see Vol. 1, White)
							· In F: gradual increase up to BMI 29-30, drop with BMI > 31 +	· Age stronger predictor of diabetes in men
							· Prevalence of diabetes increases with BMI > 31 + in M: increases with BMI > 27 - 30 in F second increases with BMI 31 + in F	· BMI and age are strong predictors of diabetes in women but BMI very strong in older women
							· Prevalence of hypercholesterolemia (Chol. > 240) Significantly related to the BMI in both men and women after adjusting for age; higher in men than in women with BMI < 25; higher in women than in men with BMI > 27	· Same apply for blood glucose levels
							· Prevalence of serum uric acid level > 7.0mg/dl much higher in men than in F and increases significantly with increasing BMI	· In M, age is a significant predictor after adjustment for BMI
							· Prevalence of serum uric SGOT > 44U/L higher in men and increases with increasing BMI	· In F, both age and BMI are good predictors
								· In M, age negatively correlated
								· In F, age positively correlated
								· In F, positively correlated but BMI stronger

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Rabkin et al. Manitoba Study (Canada)	1977	Cardiovas-cular	BMI	3 983	M	15-64	· BMI associated with IHD and myocardial infarction in those less than 35 years of age after 20 years follow-up · BMI associated with sudden death in those less than 40 years of age after 16 years follow-up · No evidence of risk elevation in the lowest BMI category · BMI highly correlated with systolic BP at initial examination	· Follow-up 26 years · World War II fighter pilots · No point above which acceleration of risk could be seen
Statszewski (Poland)	1977	Breast Cancer	BMI	1 443	F	25-69	· Risk increases with increasing BMI in post-menopausal women (24 < 27 < 30 and +) . Decreases with increasing BMI in pre-menopausal women (24 > 27 > 30 and +)	

TABLE 3
DATA BANKS: Morbidity Data (Continued)

COHORT DESCRIPTION

Source	Time	Morbidity	Measurement	N	Sex	Age	Major Findings	Specifications
Medalie et al. (Israel)	1975	Diabetes	BMI	8 688	M	40+ years	· Higher incidence in 50-59 years old with a BMI > 27.0	
U.S. National Center for Health Statistics NHANES (USA)	1971-75	Hypertension	BMI	112	M		· BMI highly correlated with diastolic BP 20 and 32 years later	· Follow-up 20 and 32 years

TABLE 4
SUMMARY OF MAJOR STUDIES RELATING OVERWEIGHT/OBESITY TO

(a) HEART DISEASE AND (b) CANCER

TWO EXCELLENT SUMMARIES OF MAJOR STUDIES RELATING EXCESS WEIGHT TO CORONARY HEART DISEASE (TABLE 4(a)) AND CANCER (TABLE 4(b)) WERE PREPARED BY HUBERT (1984) AND GARFINKEL (1985) RESPECTIVELY.

(a) Findings of prospective epidemiological studies of obesity and coronary heart disease risk in men: assessment of independent risk.

*Study	Duration (yrs)	Age (yrs)	Obesity Index	No. of events	Significant findings P < 0.05				
coronary					CHD	Myocardial infarction	Coronary insufficiency	Angina pectoris	Sudden coronary death
Framingham, U.S.A.(9,10)	26	30-62	% of Metropolitan Life desirable W	636	+		+	+	+
	20	36-68	subscapular skinfolds	375	+	+	+	+	+
Los Angeles, U.S.A.(12)	15	30-39	H/ cubic root of w	16		+			
		40-49		13				+	
Minneapolis businessmen, U.S.A. (13)	20	47-57	$W/(H^2)$	60	+	+ (infarction or CHD death)			
Manitoba, Canada (11)	26	15-64 mean age = 3i	$W/(H^2)$	390					
				209					
				31					
				50				(AP alone)	
				117					+

TABLE 4 - (Cont'd)

(a) Findings of prospective epidemiological studies of obesity and coronary heart disease risk in men: assessment of independent risk.

*Study	Duration (yrs)	Age (yrs)	Obesity Index	No. of events	Significant findings P < 0.05				
					CHD	Myocardial infarction	Coronary insufficiency	Angina pectoris	Sudden coronary death
Glostrup, Danemark (18)	10	50	% of Natvig's desirable W	31		+			
Hawaii, Japanese ancestry (14,19)	2	45-64	sum of skinfolds	91	+	+		+	
	10	45-68	$W/(H^2)$	511	+	+			
Western Collaborative, U.S.A. (8)	8.5	39-49 50-59	$W/(H^2)$	257	- -				
U.S. Railroad (7)	5	40-59	sum of skinfolds	177	-				
Yugoslavia, urban men only (20)	7	35-62	W/H	86	-	(infarction or CHD death)			
Oslo, Norway (21)	10	40-49	W/H	209	-				

CHD = coronary heart disease; H = height; W = weight; + = yes; - = no.

Reproduced, with permission, from: Hubert, H. The nature of the relationship between obesity and cardiovascular disease. Intern. J. Cardiol. 1984; 6:268-274.

TABLE 4 - (Cont'd)

(b) Relation of Overweight and Cancer in Case-Control Studies

Author, Year *(Reference)	Site	Index		Odds Ratio
		Quetelet**	Other	
Helmrich et al., 1983 (1)	Breast	> 40 vs < 30		1.30
Brinton et al., 1983 (2)	Breast	≥ 26 vs < 22		1.27
De Waard et al., 1974 (3)	Breast	≥ 29 vs < 25		1.25
Statszewski, 1977 (4)	Breast	> 30 vs < 24		0.67
Wynder et al., 1978 (5)	Breast	17-27 (premenopausal) vs 0-12 (postmenopausal)		0.63, 0.93
Wynder et al., 1966 (6)	Endometrium		10 + lb overweight vs average weight	6.69
Lavecchia et al., 1982(7)	Endometrium	≥ 30 vs < 20		7.20
Lavecchia et al., 1982(8)	Endometrium	> 25 vs < 25		3.00
Cramer et al., 1984 (9)	Ovary	> 29 vs < 22		1.53
Snowdon et al., 1984(10)	Prostate		130 % desirable weight vs 90% to 109% weight index	

* For further information, readers are referred to the respective author of the summary table.
** Body mass index

Reproduced, with permission, from: Garfinkel, L., Overweight and Cancer. Ann.Int.Med., 1985; 103(6):1034-1037.

TABLE 5

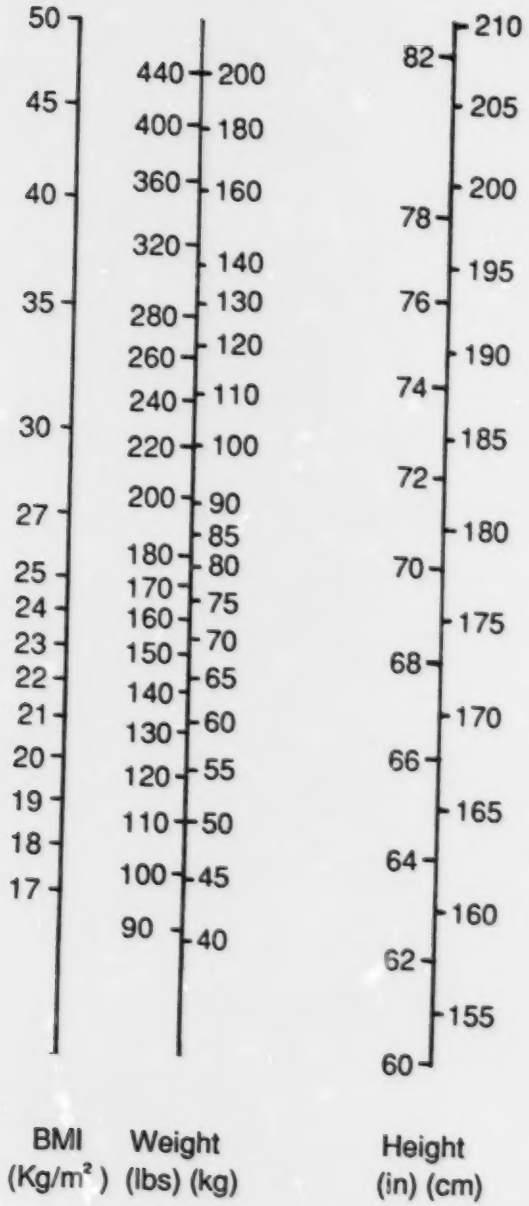
PERCENTAGE OF CANADIANS BY BODY MASS INDEX ZONES FOR HEALTHY WEIGHTS

AGE GROUP/SEX	Sample Size		ZONE A BMI < 20		ZONE B BMI: 20 - 25		ZONE C BMI: 25 - 27		ZONE D BMI > 27	
	CFS*	CHS**	CFS	CHS	CFS	CHS	CFS	CHS	CFS	CHS
Total Population										
20-69 years	10 725	3822	10.5	8.1	50.0	44.8	15.9	16.2	23.7	31.1
20-29	3411	1000	18.1	12.8	58.0	56.7	11.2	12.5	12.8	18.2
30-39	2974	793	9.7	10.1	53.3	49.4	16.2	15.6	20.9	24.8
40-49	1886	660	6.3	5.0	44.9	41.4	18.0	18.0	30.8	35.6
50-59	1465	649	3.9	4.3	41.1	33.9	19.9	19.7	35.2	42.1
60-69	989	520	4.3	5.4	35.9	31.2	20.9	17.7	38.9	45.8
Men										
20-69 years	5039	1623	4.8	4.8	45.9	42.0	20.5	19.5	28.8	33.8
20-29	1639	460	7.4	6.1	58.6	55.2	16.3	16.1	17.7	22.6
30-39	1406	345	4.7	4.6	45.9	43.2	22.3	20.3	27.1	31.9
40-49	889	289	2.6	4.2	36.1	36.7	22.9	20.4	38.4	38.8
50-59	648	287	3.2	2.4	35.7	32.8	22.8	22.3	38.3	42.5
60-69	455	242	2.9	6.2	33.4	32.2	22.2	20.3	41.5	41.3
Women										
20-69 years	5686	1999	15.4	10.9	53.7	46.7	11.7	13.6	19.1	28.9
20-29	1772	540	28.0	18.2	57.3	58.0	6.5	9.4	8.2	14.4
30-39	1568	448	14.1	14.3	60.0	54.2	10.7	12.1	15.3	19.4
40-49	997	371	9.8	5.7	52.7	45.0	13.6	16.2	24.1	33.2
50-59	817	362	4.4	5.8	45.1	34.8	17.5	17.7	32.7	41.7
60-69	534	278	5.4	4.7	38.0	30.2	19.8	15.5	36.7	49.6

* CFS = Canada Fitness Survey (Fitness Canada, 1981)

** CHS = Canada Health Survey (Health and Welfare Canada, 1978)

FIGURE 2
SAMPLE NOMOGRAM



Instructions: To obtain the BMI, identify the appropriate points on the height and weight scales and extend a straight line though these points.

Source: Dr. Barry Garner, Department of Community Health and Epidemiology, Dalhousie University

Appendices

APPENDIX I

EPIDEMIOLOGICAL TABULATIONS OF THE BODY MASS INDEX IN ASSOCIATION WITH SELECTED MORBIDITY AND BLOOD CHEMISTRY INDICATORS, BY AGE, SEX AND REGION[1]
VOLUME I

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1 A descriptive analysis and discussion based on data from the Canada Fitness and Canada Health Surveys, for presentation to a consensus meeting on weight standards.

Submitted in accordance with an agreement with the Minister of National Health and Welfare, authorized by Dalhousie University on October 17, 1985.

Halifax
December 19, 1985

Appendix I

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I. INTRODUCTION

This is the first of a two-volume working report prepared in support of a consensus meeting on the development of weight standards. The two volumes are viewed as exhibits to be the subject of an oral presentation by one of the authors, Dr. Frank White, to take place at the consensus meeting.

The present volume explicitly addresses the following terms of reference, as specified in an agreement with the Minister of National Health and Welfare, authorized by Dalhousie University on October 17, 1985:

> Epidemiological tabulations by relevant age and sex groups of the association between overweight (using the Body Mass Index) and the prevalence of hypertension and reported diabetes based on data from the Canada Fitness Survey and the Canada Health Survey.
>
> Epidemiological tabulations of Body Mass Index prevalence in Canada, for several levels of BMI, by age, sex and region based on the Canada Fitness and Canada Health Surveys.

Discussions of these tabulations is included within the body of this report, and is intended to serve as an introduction. More detailed discussions of implications for public health in Canada and for the setting of weight standards, is understood to be the main topic of the consensus meeting itself, and will be addressed at that time.

This volume also includes additional descriptive analyses not requested in the agreement, but considered to be of value for the consensus discussion: Body Mass Index and elevations in blood cholesterol, uric acid, glucose and glutamic oxaloacetic transminase (SGOT). Volume two will focus on selected studies from the scientific literature that appear to be most useful in relating actual levels of overweight (using the Body Mass Index) to specified morbidity and mortality outcomes.

II. DATABASES

Both the Canada Fitness Survey (CFS) and the Canada Health Survey (CHS) provide cross-sectional data on health indices and height/weight measurements for men and women in five regions of Canada.

Before drawing conclusions from an analysis of these data, it is important to take into account the limitations and possible biases in these population samples. Among those aged 7-69 in the CFS sample, 58% agreed to physical measurements. The CHS had an estimated 60% response rate from those aged 15+ who were asked to provide anthropometric data. If the individuals who refused to participate had reasons relating to ill health or excess weight, the inferences based on those consenting could be biased. We should expect those with excessive weight and health problems to be under-represented in these samples.

Canada Health Survey

The CHS data exhibit a serious measurement bias in view of limits set on height and weight. Using percentiles as a guide, extreme measurements were grouped with cutoff values. For men, maximum values were six feet, one inch and 229 pounds and minimum values were five feet, two inches and 121 pounds. For women, maximum values were five feet, seven inches, 202 pounds and minimum values were four feet, ten inches and 99 pounds. All men and women with more extreme measurements were represented at these cutoff values on the data file.

The effects of this arbitrary decision can be illustrated. For example, one man six feet tall weighing 225 pounds and another six feet weighing 250 pounds would end up with a similar Body Mass Index (BMI) after the calculation weight-height.[2] Similarly, a woman 160 pounds who is five feet, six inches tall and another weighing 160 pounds who is five feet, ten inches tall would have nearly equal BMIs. Consequently, calculation of the BMI based on these altered data would provide distorted figures for many individuals.

In order to make the data as accurate as possible, all those coded with cutoff figures were eliminated by us from the standard data tape provided by CHS. Although the remaining individuals on the file are correctly represented in terms of weight and height, the data cannot provide reliable prevalence rates for Canadians at the extremes of the BMI. Furthermore, this biased exclusion of 385 people with either very high or very low measurements from the 3622 men and women 20-69 who provided physical measurements causes an analysis of the association of the BMI with health data to be of questionable reliability.

Canada Fitness Survey

The CFS data can be expected to provide more accurate prevalence rates for Canadians at various BMI levels not only because the data on height and weight are of higher quality but also because there are more data. The CFS includes 15 723 men and women aged 20-69 with height and weight measurements. However, the only health measurement taken was blood pressure. Therefore, with some reservations, the CHS data was used to examine the association of the BMI with diabetes and other health indices.

III. HYPERTENSION

Prevalence rates of both diastolic and systolic hypertension at increasing levels of the BMI derived from both CFS and CHS data are displayed in Figures 1 through 8. (The BMI is calculated by the formula weight/height^2 for both men and women in the data presented). For men in the CFS, the prevalence of diastolic hypertension ($\geq$ 90mm Hg) increases with each increase of the BMI (Figure 1). At the BMI level 27-28, the rate increases to 32% from 21% at 25-26. For BMI levels 29+, the rate is 40%. Systolic hypertension ($\geq$ 140 mm Hg) in men has a similar pattern: the greatest increase occurs at the 27-28 BMI level (Figure 2).

Prevalence rates of diastolic hypertension are generally lower for women than for men. Although the CFS data show only gradual increases at each level of the BMI up to 29-30, the rate jumps from 25% to 38% at 31+ (Figure 1). The pattern is similar for systolic hypertension in women (Figure 2).

Hypertension prevalence at increasing levels of the BMI found in the CHS data does not follow a logical pattern. Rates for both diastolic and systolic hypertension in men increase gradually up to the BMI level 25-26. The drop in prevalence at 27-28 and large increase at 29-30 are difficult to understand (Figures 3 and 4). CHS data for women show increases for both diastolic and systolic hypertension up to a BMI of 29-30 and a drop at 31+. This is the opposite of the trend found in the CFS data.

Because of (a) the flaws in the CHS measurement data, (b) the larger size of the CFS sample, and (c) the more logical pattern in the CFS data of consistent increases in hypertension prevalence with increases in the BMI, further analysis of the relationship of these two variables in different age groups will be based on the CFS data.

Table 1 displays the numbers of men and women in five age groups at seven levels of the BMI for both surveys. Even if the CHS data had proven to be reliable, the sample size was too small to be broken into these age groupings.

The relationship between the BMI and both diastolic and systolic hypertension was examined in each of the five age groups of the CFS data. For men aged 20-29 and 50-59, there is a sharp increase in diastolic hypertension at the BMI level 27-28 (Figure 9). For men aged 30-49 and 60-69, the greatest increase is at the BMI level of 29-30. Fifty percent of men aged 40-69 with a BMI 29-30 had diastolic hypertension. The decrease in hypertension prevalence in men aged 50-59 with a BMI 31+ can be explained by the greater likelihood that hypertension would be detected and treated in these age groups. The pattern of increases in systolic

hypertension with increases in the BMI is much more erratic (Figure 10). Men 50-69 have much higher rates of systolic hypertension at levels of the BMI above 21-22, than younger men. For men 40-49, the peak prevalence is at the BMI level 29-30.

For women aged 20-49, the prevalence of diastolic hypertension does not show significant increase with increases in the BMI up to the 29-30 level, but there is a definite rise in prevalence at 31+ (Figure 11). Women 50-59 show a notable rise in diastolic hypertension prevalence at the BMI level 29-30. Women 60-69 have a more erratic pattern which peaks at the BMI level 27-28. Medication taken by some in this age group may explain the irregular pattern of increases. Systolic hypertension in women is highest in those with a BMI 31+, but there are steady increases beginning at the BMI level 27-28 in all women aged 30-69 (Figure 12).

Multiple regression analysis was employed to determine the strength of the association of the BMI with hypertension after controlling for the effect of age (Tables 3 and 4). The waist to hip girth ratio (WHR) was also included in this analysis as a measure of weight distribution. For both men and women, age and the BMI are independent predictors of diastolic hypertension. For men, the BMI is a stronger predictor of hypertension than age: for women, age is the more powerful predictor. The WHR is less important than the BMI, but it is a contributing factor in diastolic hypertension. There is a small but significant interaction effect between age and the BMI indicating a greater effect of the BMI on blood pressure in older age groups. The quadratic term of the BMI has a significant but negative correlation to diastolic hypertension in men. This is due to the tapering off of hypertension prevalence among those with a BMI 31+ which is described in Figure 9. For women, the quadratic term is positively correlated with hypertension prevalence describing the sharp increase at the highest levels of the BMI (Figure 11).

The regression analysis of systolic hypertension produced similar results (Table 4). Age is more highly correlated with systolic than diastolic hypertension in both men and women. The interaction of age and the BMI has a greater effect on systolic than diastolic hypertension in women. The insignificance of the quadratic term indicates that the BMI has a more linear relationship with systolic than with diastolic hypertension.

IV. DIABETES

The CHS data provide a 1.9% overall prevalence rate of diabetes in men and 3.0% in women (Table 5). The prevalence of diabetes in men increases only at a BMI level of 31+ where it is 7.8%. For women, diabetes prevalence increases at a BMI 27-30 to 6.4% and at 31+, it is 13.8%. The association between the BMI and diabetes is much stronger in women than in men (Table 6). A regression analysis was used to evaluate the effect of the BMI after adjusting for the effect of age. For men, age is a much stronger predictor of diabetes than the BMI. The BMI was found to have a non-linear association with diabetes prevalence in men which is evident from the pattern of increased prevalence only at a very high BMI. For women, age and BMI are both strong predictors of diabetes. Furthermore, there is a very strong interaction of age and the BMI indicating a stronger association of the BMI and diabetes prevalence in older women.

V. BLOOD CHEMISTRY

Cholesterol

Cholesterol levels for the CHS sample of Canadians are very high. The mean for all men and women tested was 207 mg/dl. Figure 16 displays the percent with cholesterol above 240 at increasing levels of the BMI. At lower BMI levels, men have a higher prevalence of hypercholesteremia than women, but at a BMI 27+, women have the higher prevalence rate.

The effect of the BMI on serum cholesterol was evaluated after adjusting for the effect of age in a multiple regression analysis (Table 7). There is a strong correlation between increasing age and increasing serum cholesterol, and after adjusting for age, serum cholesterol is significantly related to the BMI in both men and women. There is also a fairly significant negative interaction between age and the BMI as well as a negative quadratic effect of the BMI; the serum cholesterol level appears to drop away from its linear increase as the BMI increases to the higher levels in older men and women. This finding may be a result of the cutoffs employed in height and weight measurements previously mentioned.

Uric Acid

The distribution of men and women whose serum uric acid level was greater than 7.0 mg/dl is presented in Figure 17. Men have a much higher prevalence of abnormal uric acid values than women in this sample. The relationship between the BMI and serum uric acid level is highly significant and linear. In men, age is a significant predictor only after adjusting for the effect of the BMI (Table 8). The association with age is negative as is the interaction term which suggests that after an adjustment is made for the BMI which is higher in older men, young men are more likely to have a high uric acid level. For women, both age and the BMI have a strong positive correlation with uric acid levels.

SGOT

The distribution of men and women with serum SGOT greater than 44 U/L is presented in Figure 18. The overall prevalence of abnormal levels is greater in men than in women. For both there is a linear relationship between increasing SGOT and increasing BMI. As with serum uric acid in men, age is negatively correlated. Regression analysis indicates that younger men are more likely to have higher levels and the BMI is more closely associated with higher SGOT levels in younger men. Age is positively correlated with SGOT blood levels in women, but the BMI has a stronger effect on blood levels of younger women.

Glucose

The distribution of men and women with serum glucose above 99 mg/dl at increasing levels of the BMI is presented in Figure 19. For both men and women serum glucose increases in a linear pattern as both age and the BMI increase. There is a positive interaction between age and the BMI which indicates that the association of the BMI and serum glucose is greater in older men and women.

VI. DISTRIBUTION OF MEN AND WOMEN AT INCREASING LEVELS OF THE BMI

The BMI has been divided into seven categories ranging from <21 to 31+. The distribution of men and women in these categories has been calculated for five age groups using CFS data (Tables 11 and 12) and CHS data (Tables 13 and 14). As has been previously discussed, the CFS data should more closely represent the true distribution of Canadian men and women at these BMI levels.

In order to determine the prevalence of those at higher than average BMI levels, the cumulative percent (or prevalence) of those above four BMI cutoff values has been calculated for the CFS sample (Tables 15 and 16) and the CHS sample (Tables 17 and 18). The prevalence of those above a BMI of 35 is obviously inaccurate for the CHS data because of the arbitrary limits set on weight and height values in the data file. Otherwise, there is not a great difference between the two surveys in the prevalence of men with a BMI above 26, 28 and 30 in five age groups. Prevalence rates for women above these BMI cutoff values are higher in the CHS than in the CFS. These differences may be partly attributed to the extremely low maximum height measurement (5 feet, 7 inches) permitted for women in the CHS data. It is also possible that heavier women would be

more likely to refuse to participate in a fitness survey than in a health survey.

The distribution of men and women in the seven BMI categories have also been calculated for five regions in Canada using the CFS data (Tables 19 and 20) and the CHS data (Tables 21 and 22). The cumulative percent of those above the four BMI cutoff values has also been calculated for men and women in the five Canadian regions using both databases (Tables 23-26). The sample population from the Atlantic region had the highest prevalence rates at most cutoff values and British Columbia had the lowest. The prevalence of men in the CFS with a BMI above 26 and 28 was greater than the prevalence of women above these cutoff values. However, the prevalence of women with a BMI above 30 and 35 was greater than the prevalence of men. In the CHS sample, only the cumulative percent above 26 was greater for men.

Table 27 shows the weight ranges for four different heights (for men and women) that are comparable to the BMI levels used in the tables and graphs of this report. Prevalence of diastolic hypertension at these levels taken from the CFS reflect the concurrent probability associated with different BMI levels for men and women. Excess weight appears to pose a greater risk of hypertension to men than to women, in contrast to the apparently greater risk of diabetes in association with increasing levels of BMI among women.

1. Diastolic blood pressures were taken at the fourth phase in the Canada Fitness Survey. Therefore, any analysis based on CFS diastolic blood pressure data may overstate the prevalence of hypertension in comparison with other surveys which used fifth phase data.

Figure 1.

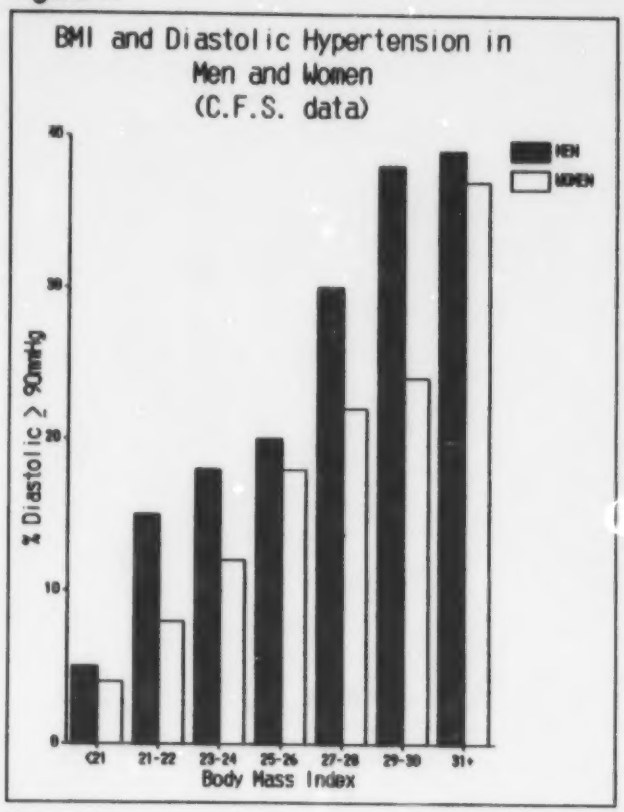


Figure 2.

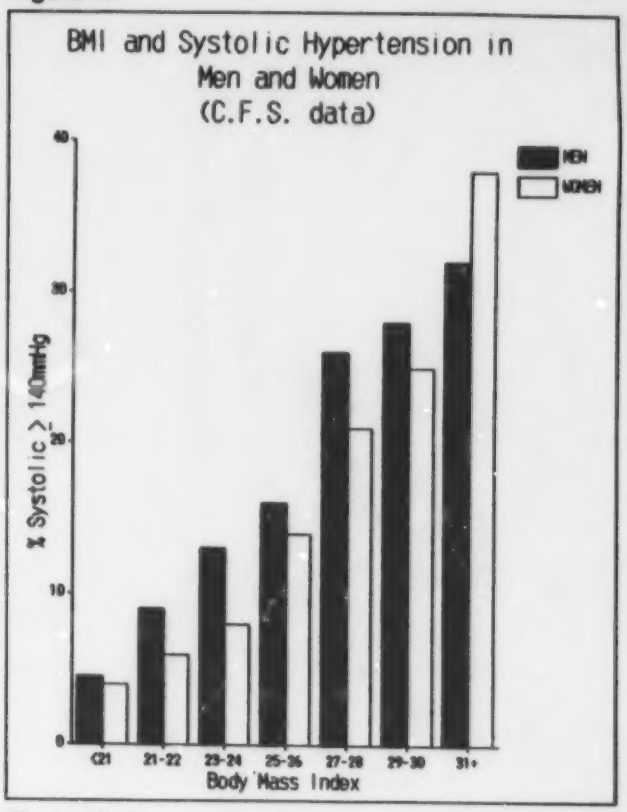
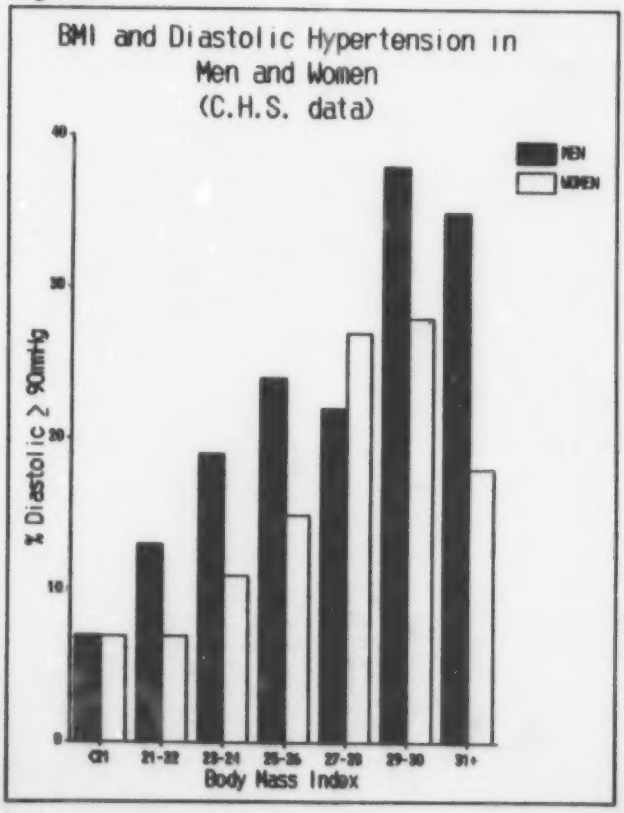


Figure 3.



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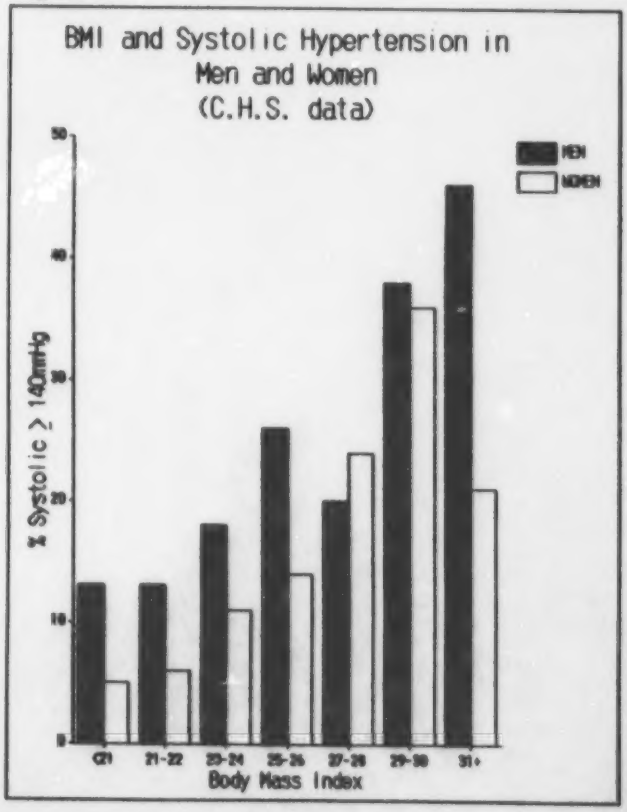

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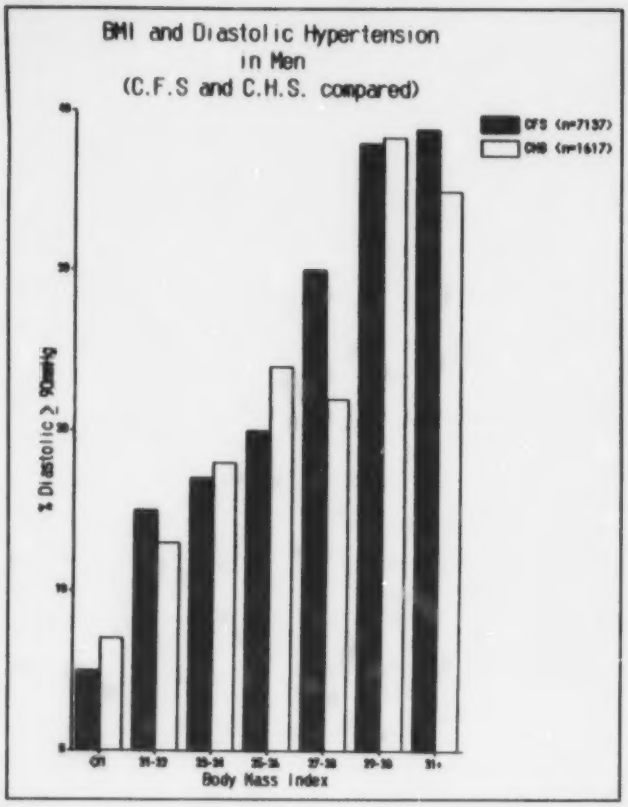
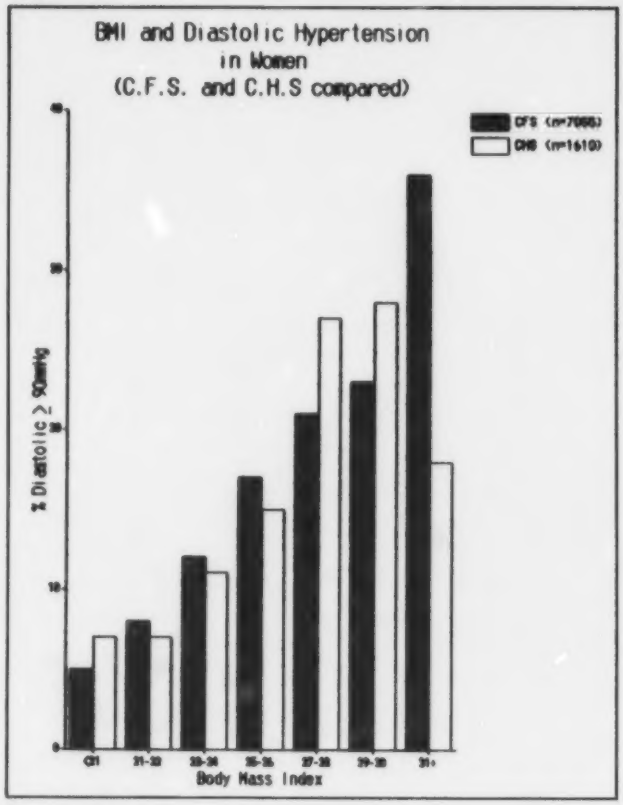


Figure 6.



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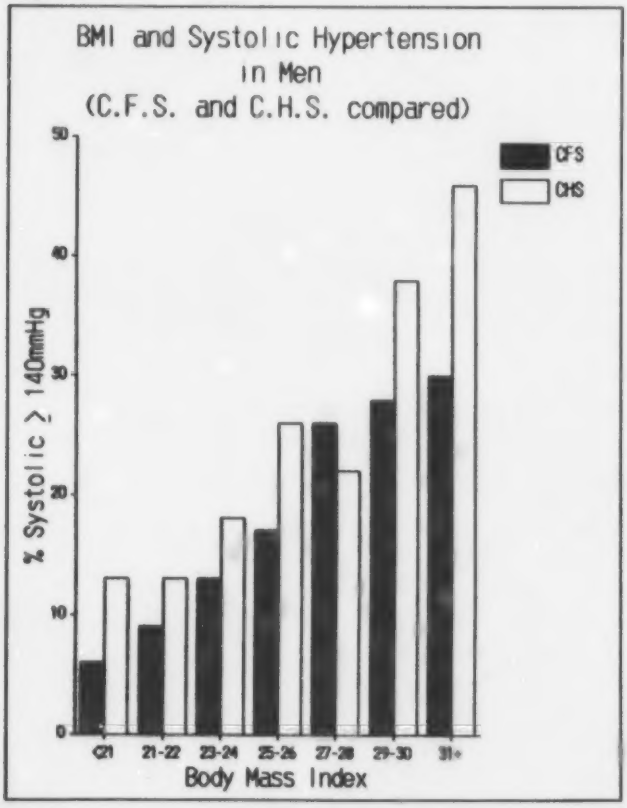

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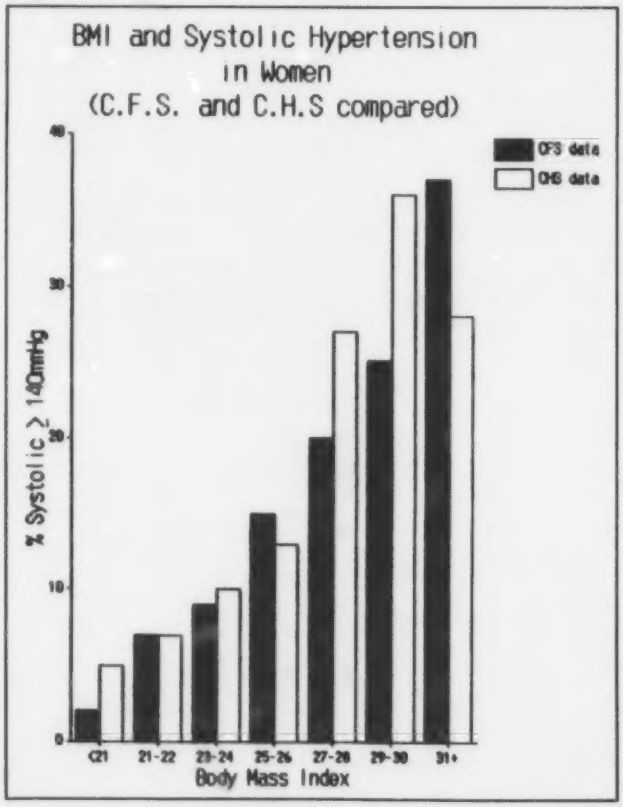

TABLE I

NUMBER IN CELLS WHEN FIVE AGE GROUPS OF MEN AND WOMEN ARE DIVIDED INTO SEVEN CATEGORIES OF BMI CFS and CHS data compared

		20-29		30-39		40-49		50-59		60-69	
BMI		men	women	men	women	men	women	men	women	men	women
<21	CFS	214	795	109	335	20	170	23	80	4	50
	CHS	74	149	12	86	8	42	12	19	12	21
21-22	CFS	584	653	284	305	134	260	103	209	63	127
	CHS	106	125	48	92	44	48	24	32	21	23
23-24	CFS	557	338	439	401	265	261	206	303	129	166
	CHS	135	86	88	89	71	48	39	59	33	23
25-26	CFS	389	173	411	209	309	166	289	217	235	207
	CHS	67	45	95	50	78	43	48	46	50	36
27-28	CFS	241	75	232	117	259	111	255	156	178	116
	CHS	74	34	68	32	48	34	80	39	33	45
29-30	CFS	117	43	184	50	149	64	145	106	86	53
	CHS	21	10	22	8	43	34	24	35	19	23
31+	CFS	77	35	126	97	123	126	100	105	100	119
	CHS	24	24	31	19	14	24	20	32	19	45

Figure 9.

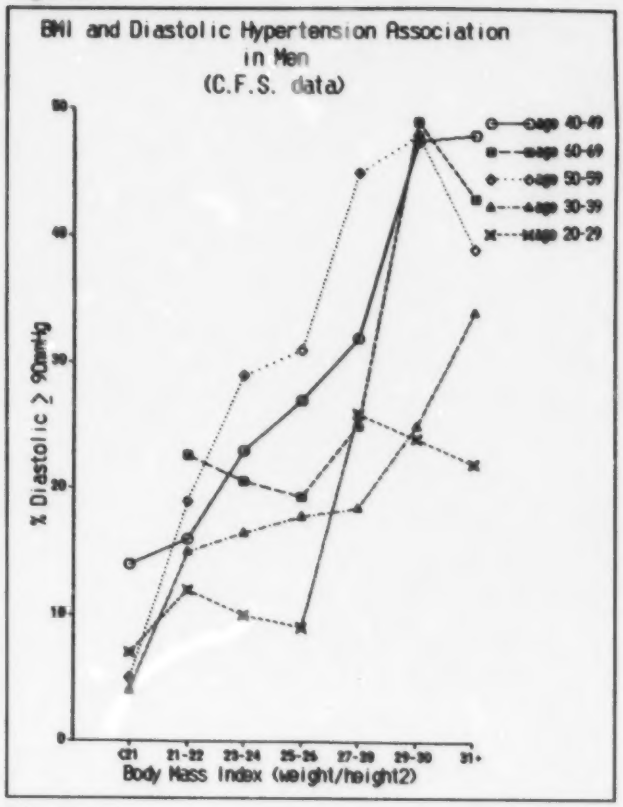

Figure 10.

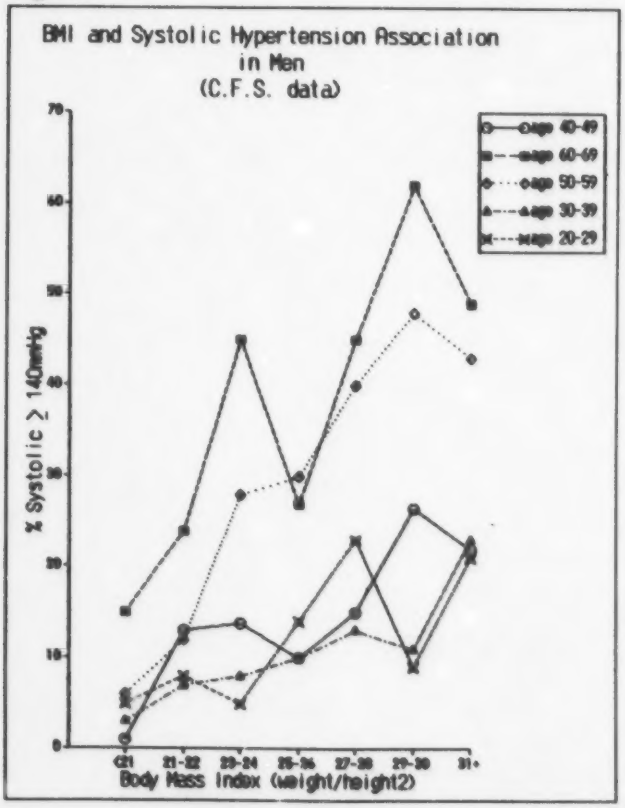

Figure 11.

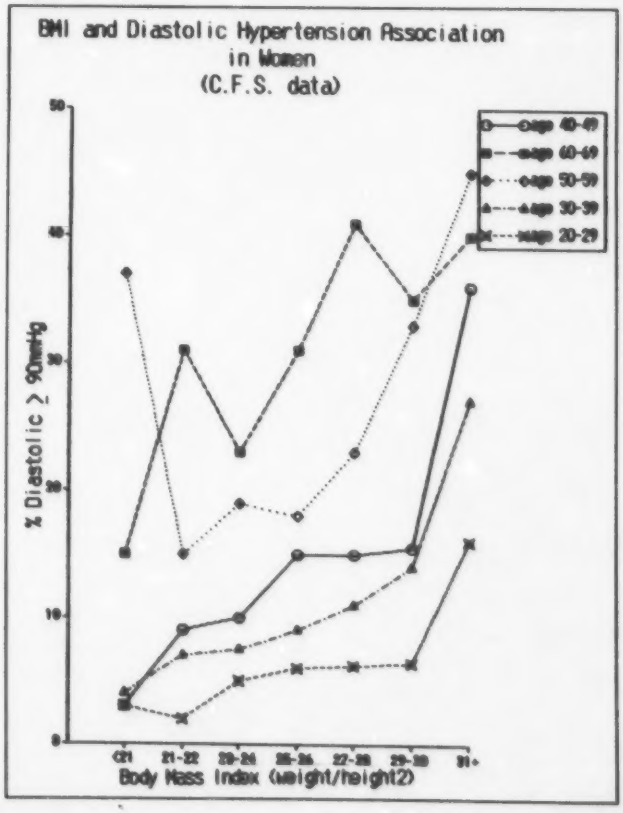

Figure 12.

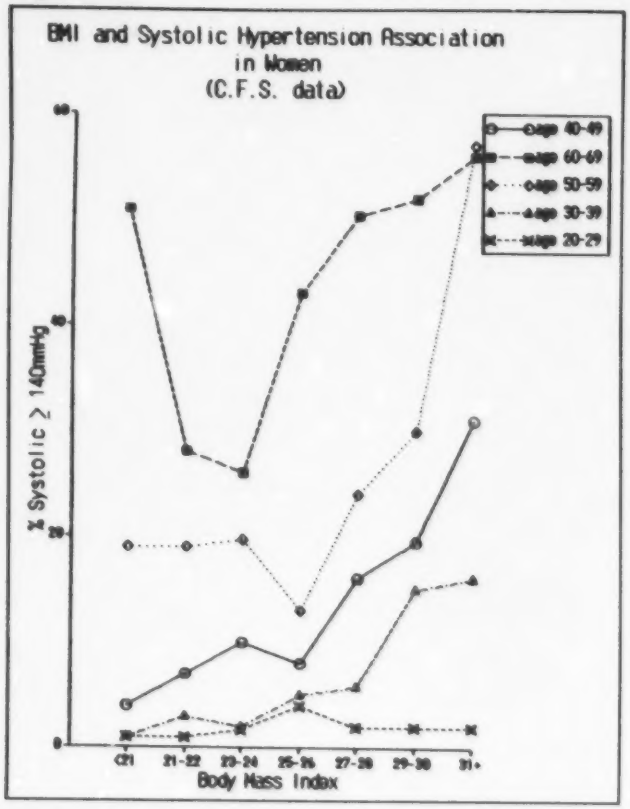

Table 2

CORRELATIONS OF BODY MASS INDEX* WITH DIASTOLIC AND SYSTOLIC HYPERTENSION IN SIX AGE GROUPS OF MEN AND WOMEN**
CFS data

AGE	MEN		WOMEN	
	DIASTOLIC	SYSTOLIC	DIASTOLIC	SYSTOLIC
20-24	.10	.15	.06	.11
25-29	.17	.15	.09	.04
30-39	.14	.12	.18	.20
40-49	.17	.18	.26	.22
50-59	.18	.21	.15	.20
60-69	.19	.17	.12	.18
ALL	.20	.20	.23	.27

*WEIGHT/HEIGHT² for both men and women
** Diastolic hypertension ≥ 90 mm Hg,
Systolic hypertension ≥ 140 mm Hg

TABLE 3

DIASTOLIC HYPERTENSION AND THE BODY MASS INDEX

CFS data

Simple and partial correlations from a multiple regression analysis of the effects of age, BMI and waist to hip girth ratio on prevalence of diastolic hypertension (≥ 90 mm Hg) (All correlations are statistically significant)

	MEN (n=6983)	WOMEN (n=6909)
AGE	.161 (.088)	.291 (.202)
BODY MASS INDEX	.207 (.107)	.239 (.120)
WAIST TO HIP GIRTH RATIO	.214 (.075)	.190 (.061)
AGE AND BMI INTERACTION	.226 (.034)	.319 (.049)
BMP	.166 (-.053)	.236 (.026)
	R=.07	R=.11

TABLE 4

SYSTOLIC HYPERTENSION AND THE BODY MASS INDEX

CFS data

Simple and partial correlations from a multiple regression analysis of the effects of age, BMI and waist to hip girth ratio on prevalence of systolic hypertension (≥ 140 mm Hg) (All correlations are statistically significant.)

	MEN (n=6983)	WOMEN (n=6909)
AGE	.274 (.199)	.405 (.325)
BODY MASS INDEX	.204 (.106)	.273 (.107)
WAIST TO HIP GIRTH RATIO	.212 (.029)	.239 (.064)
AGE AND BMI INTERACTION	.308 (.081)	.436 (.125)
BMP	N.S.	N.S.
	R=.18	R=.30

N.S. — not significant

Figure 13.

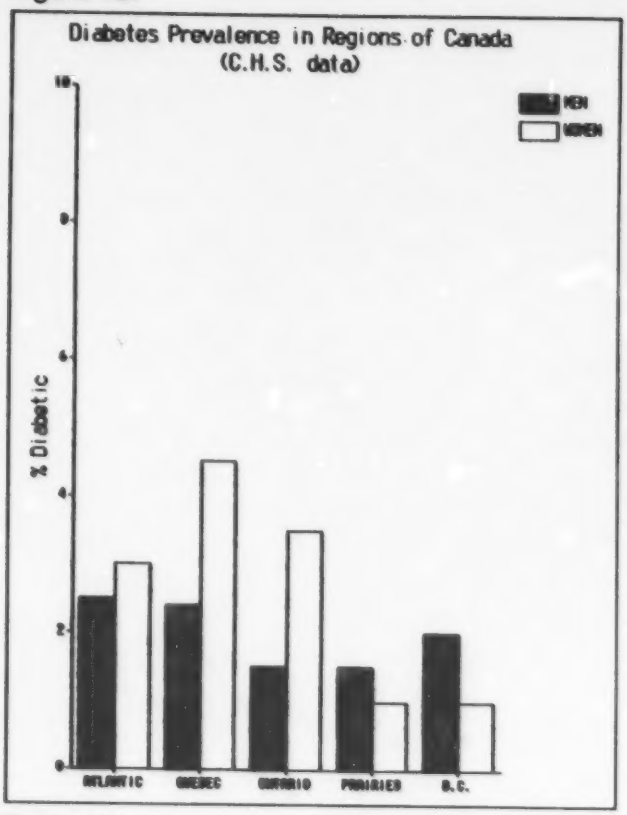

Figure 14.

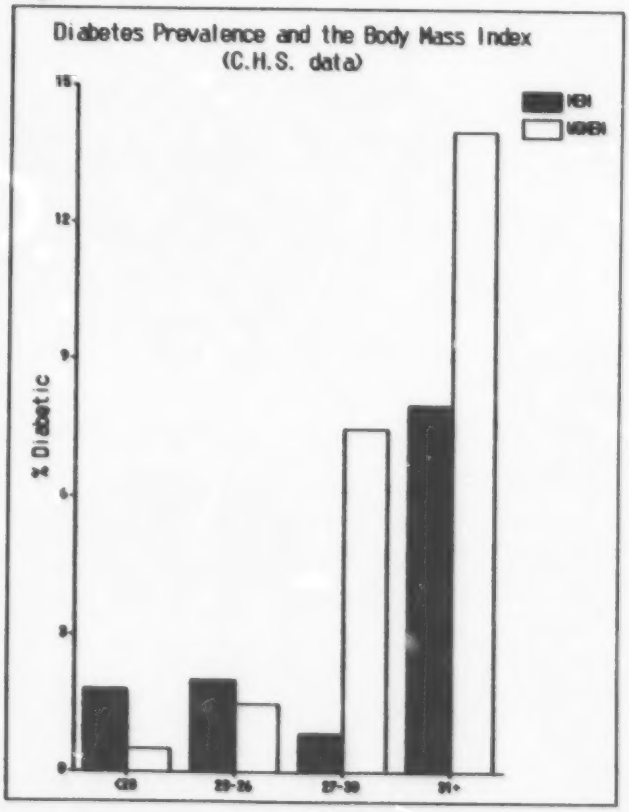

TABLE 5

PREVALENCE OF DIABETES IN MEN AND WOMEN AT DIFFERENT LEVELS OF BMI

CHS data

	<21	21-22	23-24	25-26	27-28	29-30	31+	ALL
Men n=1487	0	2.3	.5	3.5	.4	2.0	7.8	1.9
Women n=1449	.9	.1	1.4	.8	8.0	3.3	13.9	3.0

Figure 15.

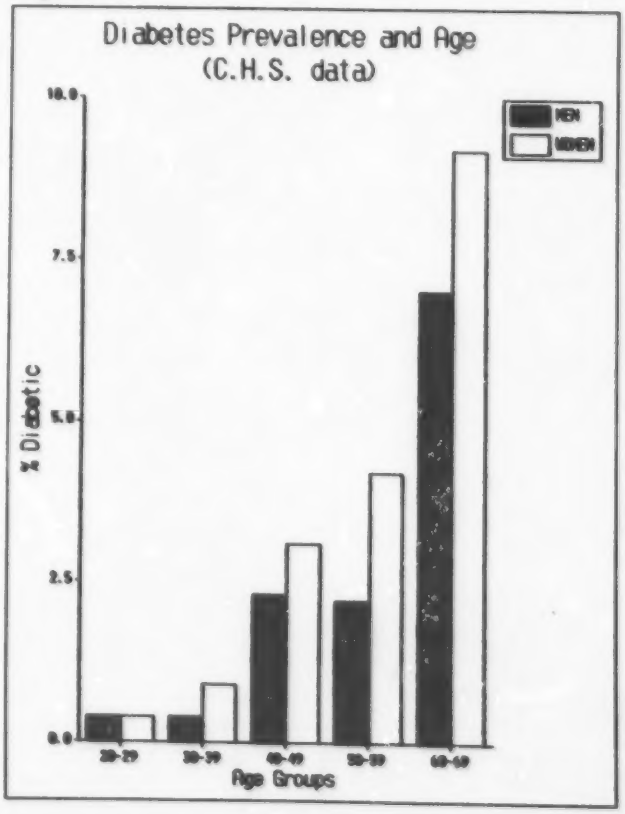

TABLE 6

DIABETES AND THE BODY MASS INDEX

CFS data

Simple and partial correlations from a multiple regression analysis of the effects of age and BMI on prevalence of diabetes.
(All correlations are statistically significant.)

	MEN (n=1487)	WOMEN (n=1449)
AGE	.149 (.135)	.204 (.144)
BODY MASS INDEX	.091 (.063)	.206 (.147)
AGE AND BMI INTERACTION	.170 (.082)	.264 (.180)
BMI^2	.101 (.119)	

Figure 16.

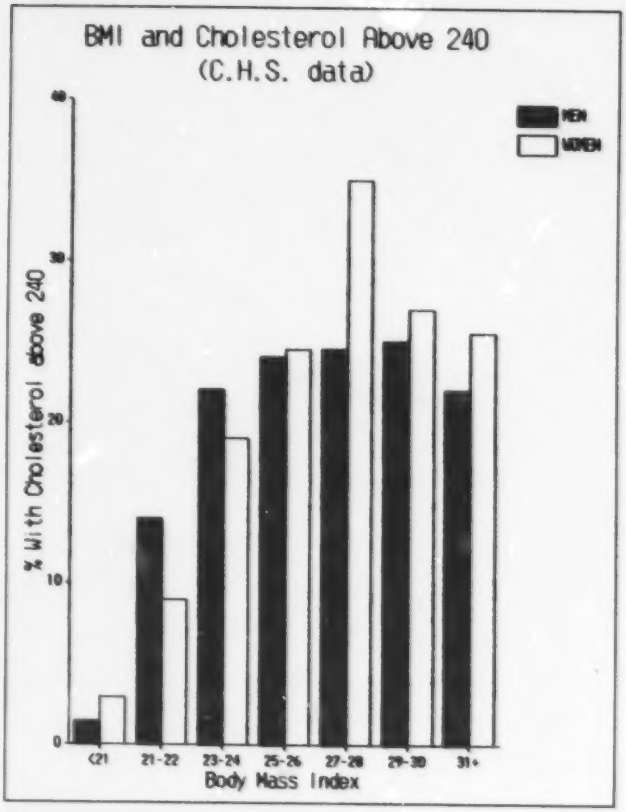

TABLE 7

CHOLESTEROL AND THE BODY MASS INDEX

CHS data

Simple and partial correlations from a multiple regression analysis of the effects of age and BMI on the level of serum cholesterol.
(All correlations are statistically significant.)

	MEN (n=1363)	WOMEN (n=1243)
AGE	.28 (.248)	.44 (.380)
BODY MASS INDEX	.24 (.191)	.30 (.179)
AGE AND BMI INTERACTION	.32 (-.072)	.45 (-.121)
BMI^2	.22 (-.137)	.29 (-.116)
	R^2=.14	R^2=.25

Figure 17.

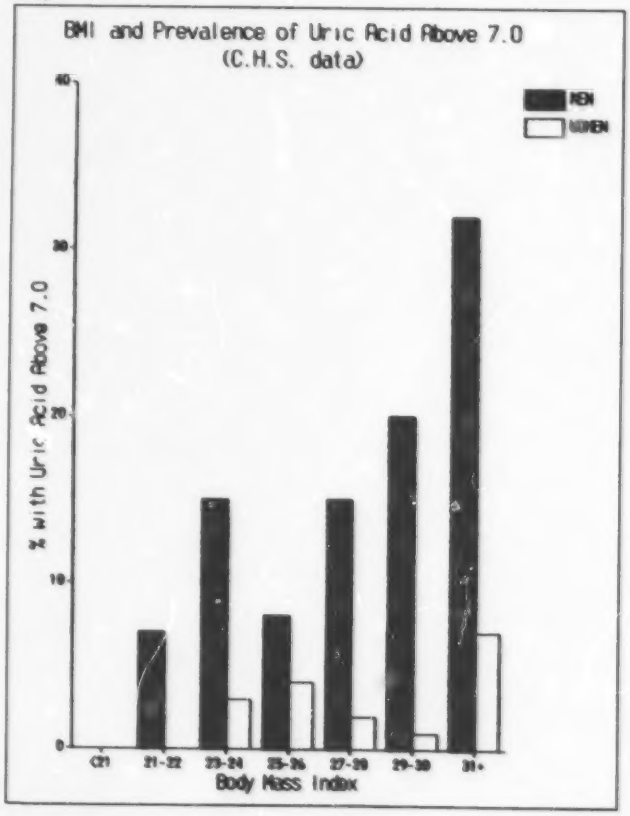

TABLE 8

URIC ACID AND THE BODY MASS INDEX

CHS data

Simple and partial correlations from a multiple regression analysis of the effects of age and BMI on the level of serum uric acid
(All correlations are statistically significant)

	MEN (n=1363)	WOMEN (n=1243)
AGE	N.S. (-.057)	.360 (.280)
BODY MASS INDEX	.274 (.279)	.320 (.224)
AGE AND BMI INTERACTION	.086 (-.084)	N.S.
BMI²	N.S.	N.S.
	R²=.09	R²=.17

TABLE 9

SGOT AND THE BODY MASS INDEX

CHS data

Simple and partial correlations from a multiple regression analysis of the effects of age and BMI on the level of serum SGOT
(All correlations are statistically significant)

	MEN (n=1363)	WOMEN (n=1243)
AGE	-.081 (-.120)	.206 (.176)
BODY MASS INDEX	.174 (.195)	.124 (.057)
AGE AND BMI INTERACTION	-.002 (-.110)	.203 (-.060)
BMI²	N.S.	N.S.
	R²=.06	R²=.05

Figure 18.

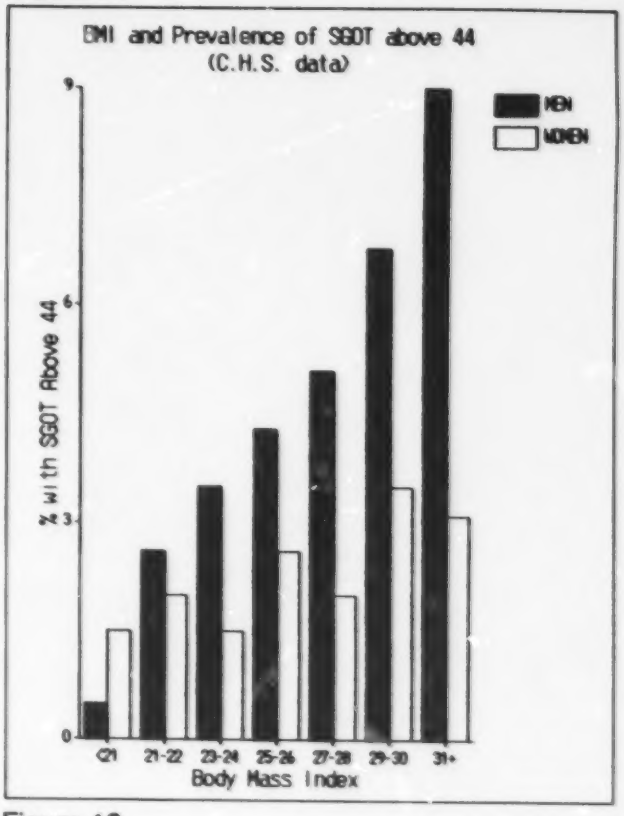

Figure 19.

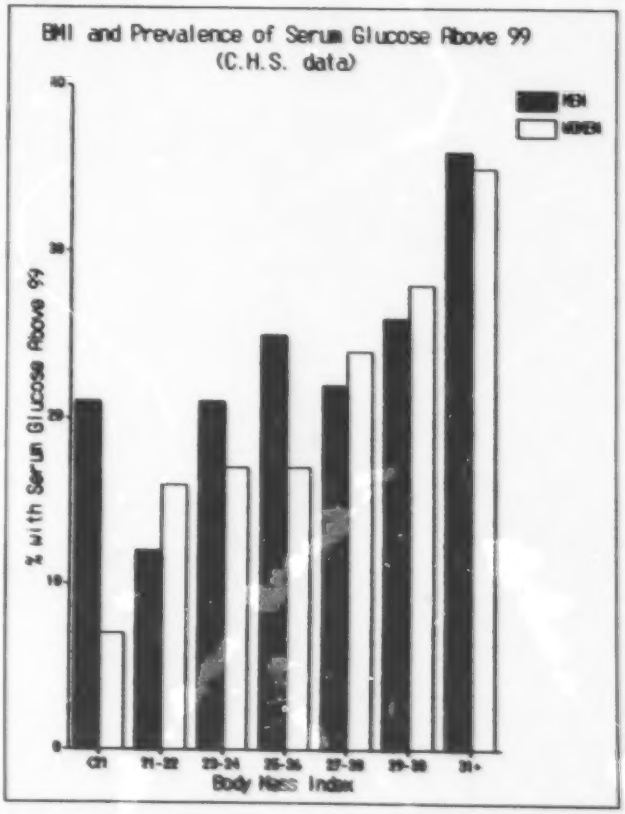

TABLE 10

GLUCOSE AND THE BODY MASS INDEX

CHS data

Simple and partial correlations from a multiple regression analysis of the effects of age and BMI on the level of serum glucose
(All correlations are statistically significant.)

	MEN (n=1363)	WOMEN (n=1243)
AGE	.246 (.230)	.258 (.201)
BODY MASS INDEX	.164 (.121)	.214 (.138)
AGE AND BMI INTERACTION	.281 (.105)	.298 (.092)
BMI^2	N.S.	N.S.
	R^2=.08	R^2=.09

TABLE 11

PERCENT DISTRIBUTION IN SEVEN CATEGORIES OF BMI FOR FIVE AGE GROUPS OF MEN

CFS data

AGE GROUPS	<21	21-22	23-24	25-26	27-28	29-30	31+
20-29	9.8	26.8	25.6	17.9	11.0	5.4	3.5
30-39	6.1	15.9	24.6	23.0	13.0	10.3	7.0
40-49	1.6	10.6	21.0	24.5	20.6	11.9	9.8
50-59	2.0	9.2	18.3	25.8	22.8	12.9	8.9
60-69	.5	7.9	16.3	29.6	22.2	10.9	12.6

TABLE 12

PERCENT DISTRIBUTION IN SEVEN CATEGORIES OF BMI FOR FIVE AGE GROUPS OF WOMEN

CFS data

AGE GROUPS	<21	21-22	23-24	25-26	27-28	29-30	31+
20-29	37.7	30.9	16.0	8.2	3.6	2.0	1.6
30-39	19.6	29.5	23.4	12.2	6.8	2.9	5.7
40-49	14.2	21.7	23.4	15.7	9.2	5.3	10.5
50-59	5.2	18.1	26.2	18.7	13.5	9.3	9.1
60-69	5.7	14.6	22.7	23.8	13.6	6.1	13.6

TABLE 13

PERCENT DISTRIBUTION IN SEVEN CATEGORIES OF BMI FOR FIVE AGE GROUPS OF MEN

CHS data

AGE GROUPS	<21	21-22	23-24	25-26	27-28	29-30	31+
20-29	14.7	21.2	27.0	13.3	14.8	4.2	4.8
30-39	3.2	13.1	26.1	25.3	18.2	5.9	8.1
40-49	2.8	14.2	23.3	25.4	15.6	14.0	4.6
50-59	4.8	9.6	16.4	19.2	32.2	9.7	8.1
60-69	6.5	11.3	17.7	26.7	17.7	10.3	9.9

TABLE 14

PERCENT DISTRIBUTION IN SEVEN CATEGORIES OF BMI FOR FIVE AGE GROUPS OF WOMEN

CHS data

AGE GROUPS	<21	21-22	23-24	25-26	27-28	29-30	31+
20-29	30.6	25.8	19.8	9.7	7.1	2.1	5.0
30-39	23.0	24.5	23.7	13.2	8.5	2.0	5.1
40-49	15.2	17.6	17.4	15.6	13.0	12.6	8.6
50-59	7.3	12.0	22.5	17.7	14.7	13.3	12.4
60-69	9.5	10.6	10.8	16.6	20.9	10.7	20.9

TABLE 15

CUMULATIVE PERCENT AT DIFFERENT LEVELS OF BMI FOR FIVE AGE GROUPS OF MEN

CFS data

BMI	20-29	30-39	40-49	50-59	60-69	ALL
above 26	19.8	30.2	42.1	44.5	45.4	33.2
above 28	8.9	17.3	21.6	21.8	24.5	16.8
above 30	3.6	7.2	9.8	9.4	12.4	7.5
above 35	.5	1.0	1.8	.5	1.2	1.0
mean	24.0	25.2	26.2	26.2	26.7	25.3

TABLE 16

CUMULATIVE PERCENT AT DIFFERENT LEVELS OF BMI FOR FIVE AGE GROUPS OF WOMEN

CFS data

BMI	20-29	30-39	40-49	50-59	60-69	ALL
above 26	7.3	16.5	25.6	33.1	34.7	20.5
above 28	3.7	9.3	16.5	19.9	20.2	12.1
above 30	1.8	6.4	10.8	10.8	14.3	7.5
above 35	.4	1.9	2.5	3.4	2.1	1.8
mean	21.8	23.5	24.7	25.6	25.7	23.8

TABLE 17

CUMULATIVE PERCENT AT DIFFERENT LEVELS OF BMI FOR FIVE AGE GROUPS OF MEN

CHS data

BMI	20-29	30-39	40-49	50-59	60-69	ALL
above 26	23.7	32.2	34.3	50.0	37.8	33.4
above 28	9.0	14.0	18.7	17.8	20.2	14.6
above 30	4.8	8.1	4.6	8.1	9.9	6.6
above 35	0	.5	.2	0	.2	.2
mean	24.0	25.5	25.5	25.9	25.6	25.1

TABLE 18

CUMULATIVE PERCENT AT DIFFERENT LEVELS OF BMI FOR FIVE AGE GROUPS OF WOMEN

CHS data

BMI	20-29	30-39	40-49	50-59	60-69	ALL
above 26	14.1	15.6	34.2	40.4	52.5	27.3
above 28	7.1	7.1	21.2	25.7	31.6	15.8
above 30	5.0	5.1	8.6	12.4	20.9	9.0
above 35	.1	.4	1.5	.2	1.1	.6
mean	22.6	23.2	24.9	25.8	26.5	24.2

TABLE 19

PERCENT DISTRIBUTION IN SEVEN CATEGORIES OF BMI IN MEN FOR EACH REGION OF CANADA CFS data

	<21	21-22	23-24	25-26	27-28	29-30	31+
ATLANTIC (n=636)	4.4	14.4	17.8	24.9	18.7	12.0	7.7
QUEBEC (n=1981)	6.0	17.9	21.8	21.7	16.8	7.8	8.0
ONTARIO (n=2633)	4.7	15.3	21.4	24.0	15.4	11.0	8.2
PRAIRIES (n=1284)	4.0	17.1	24.4	23.2	16.0	7.1	7.4
B. C. (n=850)	7.2	15.5	26.4	21.6	15.7	9.1	4.4

TABLE 20

PERCENT DISTRIBUTION IN SEVEN CATEGORIES OF BMI IN WOMEN FOR EACH REGION OF CANADA CFS data

	<21	21-22	23-24	25-26	27-28	29-30	31+
ATLANTIC (n=630)	16.0	21.0	20.7	14.7	10.1	7.5	10.0
QUEBEC (n=2022)	22.5	22.4	18.4	17.5	7.7	4.8	6.7
ONTARIO (n=2686)	17.6	24.4	22.7	14.6	8.8	3.6	8.3
PRAIRIES (n=1232)	18.8	27.5	21.9	11.6	7.4	5.6	7.2
B. C. (n=832)	22.7	27.0	23.1	9.2	9.2	2.8	6.0

TABLE 21

PERCENT DISTRIBUTION IN SEVEN CATEGORIES OF BMI IN MEN FOR EACH REGION OF CANADA CHS data

	<21	21-22	23-24	25-26	27-28	29-30	31+
ATLANTIC (n=155)	5.9	13.1	17.6	21.1	22.7	11.0	8.5
QUEBEC (n=486)	7.4	19.4	23.3	19.2	17.8	7.9	4.9
ONTARIO (n=654)	7.2	13.5	25.0	22.0	18.4	6.5	7.5
PRAIRIES (n=289)	5.7	11.5	23.3	19.9	21.3	11.3	7.0
B. C. (n=197)	11.0	16.1	22.7	22.2	15.6	6.3	6.1

TABLE 22

PERCENT DISTRIBUTION IN SEVEN CATEGORIES OF BMI IN WOMEN FOR EACH REGION OF CANADA CHS data

	<21	21-22	23-24	25-26	27-28	29-30	31+
ATLANTIC (n=160)	12.5	21.4	17.7	12.6	14.7	9.0	12.1
QUEBEC (n=519)	16.8	17.9	22.5	10.8	14.9	8.3	8.9
ONTARIO (n=670)	22.3	16.9	18.2	15.8	10.1	6.7	10.1
PRAIRIES (n=291)	22.1	22.7	20.7	12.4	9.5	5.8	6.8
B. C. (n=201)	19.3	28.3	17.0	16.8	8.9	3.1	6.1

TABLE 23

CUMULATIVE PERCENT AT DIFFERENT LEVELS OF BMI IN MEN FOR EACH REGION OF CANADA

CFS data

BMI	Atlantic	Quebec	Ontario	Prairies	B. C.	All
above 26	38.5	32.6	34.5	33.3	29.2	33.2
above 28	19.8	15.8	19.1	14.5	13.5	16.8
above 30	7.7	8.0	8.2	7.4	4.4	7.5
above 35	.6	.8	1.3	1.0	.6	1.0
mean	25.7	25.2	25.5	25.2	24.9	25.3

TABLE 24

CUMULATIVE PERCENT AT DIFFERENT LEVELS OF BMI IN WOMEN FOR EACH REGION OF CANADA

CFS data

BMI	Atlantic	Quebec	Ontario	Prairies	B. C.	All
above 26	27.6	19.2	20.7	20.2	18.1	20.5
above 28	17.5	11.5	11.9	12.8	8.8	12.1
above 30	10.0	6.7	8.3	7.2	6.0	7.5
above 35	2.1	.8	2.6	1.4	.3	1.8
mean	24.5	23.7	24.1	23.7	23.4	23.8

TABLE 25

CUMULATIVE PERCENT AT DIFFERENT LEVELS OF BMI IN MEN FOR EACH REGION OF CANADA

CHS Data

BMI	Atlantic	Quebec	Ontario	Prairies	B. C.	All
above 26	42.2	30.6	32.2	39.6	27.9	33.4
above 28	19.5	12.8	13.9	18.3	12.4	14.6
above 30	8.5	4.9	7.5	7.0	6.1	6.6
above 35	0	.1	.3	.2	0	.2
mean	25.7	24.8	25.1	25.6	24.6	25.1

TABLE 26

CUMULATIVE PERCENT AT DIFFERENT LEVELS OF BMI IN WOMEN FOR EACH REGION OF CANADA

CHS Data

BMI	Atlantic	Quebec	Ontario	Prairies	B. C.	All
above 26	35.8	32.1	26.8	22.2	18.8	27.3
above 28	21.1	17.2	16.8	12.6	9.9	15.8
above 30	12.1	8.9	10.1	6.8	6.1	9.0
above 35	.2	1.0	.3	.3	.5	.6
mean	24.9	24.5	24.2	23.7	23.5	24.2

TABLE 27

WEIGHT RANGES FOR MEN AND WOMEN AT 4 HEIGHTS COMPARABLE TO BMI CATEGORIES AND PROBABILITY OF DIASTOLIC HYPERTENSION (≥90 mm Hg)

(CFS data on Hypertension)

BMI*	5'4"	5'6"	5'8"	5'10"	Hypertension Probability	
	lbs.	lbs.	lbs.	lbs	men	women
21-22	125-132	130-138	140-148	148-155	14.8	8.1
23-24	133-142	139-151	149-162	156-170	17.3	11.9
25-26	143-154	152-163	163-174	171-185	20.1	16.9
27-28	155-165	164-176	175-187	186-200	30.1	21.8
29-30	166-179	177-189	188-204	201-215	38.1	23.7
31+	180+	190+	205+	216+	38.6	36.4
35	205	215	230	245		

* Values are rounded off (e.g., range 23-24 is actualy 22.5 to 24.4)

$BMI = \frac{\text{weight (kg)}}{\text{height}^2\text{ (m)}}$

Note: BMI categories 21-26 are consistent with acceptable weights for men according to the ranges given by the Metropolitan Life Insurance Company in 1983.

APPENDIX II

EPIDEMIOLOGICAL TABULATIONS OF THE BODY MASS INDEX IN ASSOCIATION WITH SELECTED MORBIDITY AND BLOOD CHEMISTRY INDICATORS, BY AGE, SEX AND REGION[1] VOLUME II

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1 A compilation and discussion of the selected studies from the scientific literature that appear to be most useful in relating actual levels of overweight using the Body Mass Index to specified morbidity and mortality outcomes.

Submitted in accordance with an agreement with the Minister of National Health and Welfare, authorized by Dalhousie University on October 17, 1985.

Halifax
December 24, 1985

Appendix II

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I. INTRODUCTION

This is the second of a two-volume report prepared in support of a consensus meeting on the development of weight standards. This volume derives from a scientific review of the epidemiologic literature linking weight level to total mortality, cardiovascular morbidity and mortality, hypertension, cholesterol levels, diabetes and some malignant neoplasms.

The material presented herein has been selected from the literature as the most useful in relating actual levels of overweight (using the Body Mass Index) to morbidity and mortality outcomes.

A more comprehensive but less focussed review has already been presented in accordance with an earlier contract,* and the epidemiology of overweight in Canada in reference to the development of a health promotion strategy had also been presented in the form of an earlier summary report.**

It is intended that the evidence presented in both volumes of this report, focussing on actual levels of the Body Mass Index in association with indicators of health risks and adverse outcomes, should prove to be most relevant to the development of weight standards based on this index.

II. BACKGROUND

In 1983, the Metropolitan Life Insurance Company released a new standard of acceptable weights[1] which is proposed to replace their 1959 Desirable Weight Tables.[2] Based on the 1959 Build and Blood Pressure Study (BBP),[3] the 1959 tables have been the most widely used reference in studies of the association of weight to mortality and disease. The 1959 "desirable weights" were derived from an examination of mortality experience of five million insured persons followed from 1935 to 1954.

The validity of these tables has been questioned by many[4,5,6] for a number of reasons: 1) The actual length of follow-up was determined by the date the policy was issued until cancellation of the policy, death or the end of the study. Therefore, mortality rates were based on groups of people with greatly varying lengths of follow-up. The average duration of follow-up was 6.6 years. 2) The insured population is a self-selected sample and likely to differ from a normal population in distribution of weights and general health. 3) Measurements of height and weight were not systematically taken and some were self-reported. 4) Frame size was not derived from anthropometric measurements, but was based on an arbitrary division of the population into 3 groups. 5) Policy holders in the 1959 BBP Study weighed less on the average than Americans measured in a national survey in the same time period.[7]

A comparison of the 1959 BBP Study and the 1979 Build Study[8] populations revealed that the 1979 insured men were heavier, young women weighed more, and older women weighed less than in the previous study. Overall, mortality rates were lower, particularly among those of less than average height who were mild to moderately overweight.[9] The acceptable weight ranges have increased 10% for shorter persons and 5% for

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See: * Stockwell, H., Periera L., White, F., Cottreau C., The Association between Body Weight and Leading Causes of Morbidity and Mortality: An Epidemiologic Reconnaissance, February 20, 1985.

** White F., The Epidemiology of Overweight in Canada in Reference to the Development of a Health Promotion Strategy, Summary Report, April 26, 1985.

those of moderate height in the 1983 weight tables. These changes in the Metropolitan Life Insurance Tables bring into question the validity of either build study and the usefulness of weight standards based on them.

Increasingly, researchers are selecting the Body Mass Index (BMI)* as a weight measure because 1) it is independent of the measurement of any particular population; 2) it has a low correlation with height and high correlation with more direct measures of body fat, and 3) it is simple to calculate and more precise than skinfold measurements.[10] According to Bray[11], the acceptable ranges for the BMI are 20-25 for men, 19-24 for women. Men 26-30 and women 25-30 are overweight, and both men and women with a BMI over 31 are obese. Using mortality data from the 1959 BBP study as a reference, Bray notes that "it is not until this index rises to 30 or more that there is a significant excess risk". A review of epidemiological studies which not only attempt to establish a relationship between morbidity, mortality and risk factors and the BMI, but indicate degrees of risk at specific BMI levels will help verify or disprove this generalization.

III. MORTALITY

There is considerable controversy concerning the relationship between weight and mortality. The 1959 BBP Study,[3] the 1979 Build Study,[8] and the American Cancer Society Study[12] describe increases in mortality risk for those moderately overweight. Other studies of weight and mortality include the Chicago Peoples Gas Company Study, the Seven Countries Study, the Whitehall Study, Framingham Study, and most recently, a study from Norway.

The American Cancer Society data were acquired from a questionnaire survey. All heights and weights were self-reported.

Relative weight was defined as the percent above the average weight for persons in the same age and height category (a table of average weights for their sample is included in their report). A woman aged 55 who is five feet four inches tall and weighs 138 pounds is 5% underweight by the American Cancer Society definition (weight/average weight = 138/145.7) and 15% overweight using the 1959 Metropolitan Life Insurance Tables (weight/average weight = 138/120). So the claim by the American Cancer Society Study that men and women who are between average weight and 20% below average weight experience the lowest mortality can be translated to lowest mortality among those between desirable weight and 20% over desirable weight by Metropolitan Life Insurance standards.

The Chicago Peoples Gas Company's 14-year study[13] and the Seven Countries' 10-year mortality study[14] describe high mortality rates for men at the Metropolitan Life Insurance "desirable weights", lowest mortality for those moderately overweight and high mortality for those extremely overweight. These and several other studies[15,16,17] have examined risk factors in relation to mortality (e.g. smoking, hypertension, cholesterol) and after controlling for the effects of these variables, no relationship between overall mortality and weight was found.

A recent Norwegian ten-year mortality study[18] included 1 717 515 men and women aged 20 and over. The mortality rates for 14 age groups 20-89 years at increasing levels of BMI are displayed in Figures 1 and 2. Lowest mortality for men in most age groups was associated with a BMI from 23-25. Definite increases in rates occur at 29 for some age groups and 31 for others. A U-shaped curve is most pronounced for those 30-49 years of age, showing highest mortality rates for those with BMIs $\leq$ 19 and $\geq$ 31. Among women, mortality rates are lowest for those with a BMI between 23 and 27. Rates for women

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See: * White, P., Periera L., Measurements of Obesity and Overweight in Practical Settings - A Third Report to the Minister of National Health and Welfare, for more comprehensive discussion of the BMI.

increase gradually to a BMI of 29 and the U-shaped curve is not as pronounced indicating that women have a greater tolerance for excess weight. Figures 3 and 4 display average BMIs for those who died of various causes. No adjustment for cigarette smoking, hypertension and other known risk factors was made in this study.

The Seven Countries Study[14] of mortality and coronary heart disease followed samples of men aged 40-59 in the United States, Finland, Japan, northern and southern Europe for 10 years. A significant negative relationship between the BMI and all causes of mortality was found in southern Europe and the United States. After adjusting for age, systolic blood pressure, serum cholesterol, smoking and pulse rate, the relationship between weight and mortality among a cohort of U.S. Railroad men (included in the Seven Countries Study) was curvilinear (Figure 5). Death rates were highest among those with a BMI < 22 and subsequently declined as the BMI increased up to approximately 32 at which point an increase in the probability of death can be observed. Lowest mortality was experienced by those with a BMI 26 to 32. The Chicago Peoples Gas Company Study[13] evaluated the association of the BMI with mortality in 1287 men aged 40-59 years during a 14-year period. The analysis included age, systolic blood pressure, serum cholesterol and cigarette smoking. A significant quadratic relationship was found between the BMI and all causes of death, cardiovascular renal death and coronary heart disease. Mortality was lowest among men 25 to 35% over Metropolitan Life Insurance standards. Men at the highest risk were either near Metropolitan ideal weight or 50% above it. Table 1 displays the rate/1000 deaths for three mortality end points at increasing levels of the BMI. Rates were given for all men, and smokers and non-smokers separately. In all three groups, lowest mortality was experienced by men with a BMI between 27 and 29.

In the Whitehall Study[19], 18 393 male civil servants aged 40-64 were followed for ten years. Mortality rates for six age groups at increasing levels of the BMI are shown in Figure 6. A significant linear association between the BMI and mortality rates is reported for those aged 40-49. For those 50-59, there is a significant quadratic association with the highest mortality rates for those with a BMI $\leq$ 22 and > 27. The relationship between mortality and the BMI for those 60-64 is linear and negative; the heaviest men have the lowest mortality. In examining coronary heart disease mortality (Table 2), there is a significant positive linear trend of increased mortality with increases in the BMI, particularly for those 50-59. However, after controlling for the effects of blood pressure, cholesterol and cigarette smoking, the independent effect of weight was negligible.

A study of six year mortality for women in the Framingham Study supports the other American studies.[17] Mortality rates which have been adjusted for age and other risk factors indicate lowest mortality for women with BMIs 21-31 (Table 3). Those with higher or lower BMIs have increased risk. For men, the lowest mortality is found among the heaviest group, those with BMIs 32-38. The relationship between relative weight and overall mortality, after 18 years of follow-up of the Framingham Cohort, was significant and negative for men though not significant but negative for women.[20]

IV. CARDIOVASCULAR MORBIDITY

In many studies of risk factors for cardiovascular disease, weight level has had no positive (adverse) effect on the incidence of disease after adjusting for hypertension, serum cholesterol and smoking.[13,14,21,22]

A recent report from the Framingham Heart Study[23] presented results of a 26-year follow-up of 2 252 men and 2 818 women aged 26-62 at entry. Excess risks associated with relative weight were experienced almost totally by those under 50 years of age. After adjusting for other risk factors in men, relative weight was associated with coronary heart disease, angina pectoris, sudden death

and congestive heart failure. Among women, relative weight was associated with coronary heart disease, angina pectoris, myocardial infarction, congestive heart failure and stroke.

The Manitoba Study[24] followed 3983 men aged 15-64 at entry also for a 26-year period. The BMI was associated with myocardial infarction, sudden death and coronary insufficiency or suspected myocardial infarction. When separate age groups were analyzed, it was found that the BMI was associated with ischemic heart disease and myocardial infraction only in those less than 35 years of age and after 20 years of observation. The BMI was associated with sudden death in those less than 40 years of age after 16 years of follow-up. Morbidity ratios for all manifestations of ischemic heart disease at different levels of the BMI can be found in Table 4. The BMI was most strongly related to sudden death.

V. HYPERTENSION

There is very little controversy concerning the association of weight with hypertension. Several cross-sectional and longitudinal studies have demonstrated higher prevalence rates of hypertension among heavier individuals and increases in blood pressure with weight gain.[25] Data from the U.S. National Health and Nutrition Examination Survey, 1971-1975,[26] indicate that age and BMI are independent predictors of hypertension. The relationship between BMI and hypertension is strongest in those aged 25-54, and weakest in those aged 65-74.

In a prospective study of hypertension,[27] measurements were taken from 112 college men including height, weight, various indices of body fat, diastolic and systolic blood pressure. They were examined again 20 and 32 years later. The BMI was more closely associated with systolic than diastolic blood pressure at the initial examination, whereas in middle age, the BMI was more highly correlated with the diastolic blood pressure. Weight at initial examination was significantly related to diastolic blood pressure 20 and 32 years later, but not to systolic blood pressure after the same follow-up period. Weight gain during the period of follow-up was significantly associated with increases in both diastolic and systolic blood pressure.

Although it has been established that excess weight is a risk factor for hypertension and that hypertension is a risk factor for cardiovascular mortality, excess weight has not been found to be a strong predictor of cardiovascular mortality. Two recent prospective studies have compared overall and cardiovascular mortality rates for normotensives and hypertensives at low and high levels of BMI.[28,29] Both studies found that hypertensives with a lower BMI had higher mortality rates than hypertensives with a high BMI. Normotensives had increased mortality rates with increased BMI. Table 5 compares nine-year mortality rates of those with diastolic blood pressure < 95 and ≥ 95 among 1 727 men aged 50-79. Overall mortality rates, and cardiovascular and ischemic heart disease mortality rates are displayed for hypertensives and normotensives at two levels of BMI (< 27, ≥ 27). Only among the non-obese do those with hypertension have higher mortality rates than those without. Among hypertensives, the apparently beneficial effect of excess weight indicates a possible difference in the etiology and pathology of hypertension in the obese.

VI. CHOLESTEROL

The contribution of BMI levels to cholesterol levels has not been extensively studied. In a large population survey in Tecumseh, Michigan,[30] it was found that correlations of cholesterol with relative weight were significant for men aged 15-39, and among women aged 30-39. Correlations were insignificant after the age of 40.

Table 6 displays average serum cholesterol levels for male and female blood donors aged

20-69.[31] Increases in cholesterol with increases in BMI levels are more pronounced in men aged 20-49. The rise is greatest at the BMI level $\geq$ 28.5. Cholesterol increases more gradually with increasing BMI in women.

A random sample of 107 healthy men (employees of an electronic industry near Naples) were studied to evaluate the relationship between excess weight and plasma lipoproteins.[32] Table 7 shows a comparison of the average cholesterol and triglyceride lipoprotein plasma concentrations in men in the lowest quintile of BMI (approximately $\leq$ 23) with men in the highest quintile of BMI (approximately $\geq$ 28). The conclusion drawn from this study was that the overproduction of very low density lipoproteins is probably responsible for higher plasma triglyceride and cholesterol concentrations in overweight men.

VII. DIABETES

The prevalence of maturity onset diabetes and obesity both increase markedly with age. There is no controversy over the relationship of diabetes and weight. Those with a family history may have a predisposition to diabetes and are at much greater risk,[33] although excess weight will also increase the risk of diabetes in those without a family history. Duration of obesity[34] and centrally located body fat[35] have also been identified as risk factors. Table 8 displays prevalence rates for diabetes from the 1976 U.S. National Health Interview Survey divided into age and BMI groups.[36] The prevalence of diabetes increases more rapidly with increases in the BMI among women than among men. However, in the lower weight groups, prevalence of diabetes is greater in men than among women. Table 9 describes the five-year incidence of diabetes in Israeli men with increasing BMI in three age groups.[37] Those 50-59 have a higher incidence than the younger and older men and the increase at BMI level > 27.0 is much greater.

VIII. CANCER IN WOMEN

In a case-control study of breast cancer in both premenopausal and postmenopausal women in San Francisco,[38] several risk factors were found significant for both groups: early menarche, fewer pregnancies, delay in first pregnancy, late menopause, family history of breast cancer, higher educational and occupational status. Weight measured by the BMI was significant for postmenopausal women; that is, the risk of breast cancer was evaluated at higher levels of the BMI (Table 10). However, in premenopausal women, the risk of breast cancer became lower with increasing BMI. The amount of weight gain since age 20 also significantly increased the risk of breast cancer in postmenopausal women.

Lubin et al.[39] compared women with breast cancer age 60 and over in Israel with a control group and found a similar increase in risk (odds ratio) with higher levels of BMI (Table 11). There appears to be no particular level at which increase in risk is not evident nor at which the increase levels off. In this study, adjustment was made for other known risk factors for breast cancer using logistic regression analysis. This resulted in small reductions in the odds ratios.

In a similar case-control study in Poland,[40] women aged 25-49 and women 50-69 were compared separately with matched controls (Table 12). The same pattern of decreasing risk at higher BMI levels in the younger women and increasing risk of breast cancer with higher BMI levels in older women (most of whom were probably postmenopausal) was observed. It is noteworthy that the BMI cutoffs selected for the women in San Francisco are much lower than those for the women in Poland. The highest level of BMI for the former group is 24+ and the lowest level for the latter group is < 24. Despite the differences in the chosen categories for the BMI, we can still see an increasing trend of breast cancer risk at both the lower and upper levels.

It has been suggested that in postmenopausal women, obesity is associated with adipose tissue estrogen secretion.[39] Although all other risk factors are the same for breast cancer in pre- and postmenopausal women, it is possible that the etiology for these two groups is different. An explanation given for the association of BMI with breast cancer in lean premenopausal women is that less invasive tumors are easier to detect in thin women.

In a longitudinal survey of 121 964 nurses aged 30-55 in the U.S.[41] who were followed for 4 years, lean women were found to have a higher risk of breast cancer than heavier women, but the tumors were less than 2.0 cm in diameter and were not associated with metastases.

A case-control study in Milan,[42] of 173 women with endometrial cancer and 347 control women, evaluated the risk of obesity, estrogen replacement therapy and other factors in relation to endometrial cancer. Obesity was found to be the most important risk factor (Table 13). Non-contraceptive estrogen was also associated with an increased risk of endometrial cancer, but this risk was higher in women with a BMI < 25 than in those with a BMI > 25.

IX. CANCER IN MEN

The Japan-Hawaii Cancer Study[43] included 8006 Japanese men who were examined between 1965 and 1968 and followed for 15 years. Table 14 shows a strong negative relationship between stomach cancer and the BMI. There appears to be an association of colon cancer with the BMI, but it is not significant. However, when the men were divided into two age groups (< 55, $\geq$ 55), a strong positive relationship between colon cancer and the BMI was revealed for men $\geq$ 55 years of age. Figure 7 shows the different trend of colon cancer with increasing BMI for younger and older men. A reversed J-shaped curve can be observed for stomach cancer in men in both age groups. The highest risks are in leaner men except for the highest category of BMI (26.3 - 44.6).

BMI at age 25 was also analyzed with regard to cancer incidence. Prostate cancer had a weak association with this variable (P=.06). Incidence of prostate cancer for the 5 levels of BMI at age 25 were 7.8, 12.7, 10.9, 18.9 and 15.5 per 1000 men.

Cancer incidence rates were also compared with weight gain between age 25 and time of examination. Stomach and lung cancer had significant negative relationships to weight gain (P=.015, P=.032). Colon cancer had a weak positive association with weight gain (P=.07). However, a significant relationship between weight gain and colon cancer was observed in men aged 55 or more (P=.01). Furthermore, it was found that for colon cancer, weight gain was a more important factor than a high BMI throughout life.

X. CONCLUDING COMMENT

Although excess weight has been found to be associated with hypertension, diabetes, serum cholesterol, breast, endometrial and colon cancer, it is one among several factors contributing to these diseases. Furthermore, obesity is a heterogeneous condition: that is, not all obese individuals are unhealthy.[9] We need to be able to identify those overweight people who are at increased risk of disease. There are also many individuals within commonly accepted weight "standards" who have elevated serum cholesterol, hypertension or diabetes, and some of these may benefit from weight reduction.

We also need a better understanding of the relationship between age and weight. Although there is a clear association between weight levels and disease, the association with longevity is less clear.

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Figure 1.

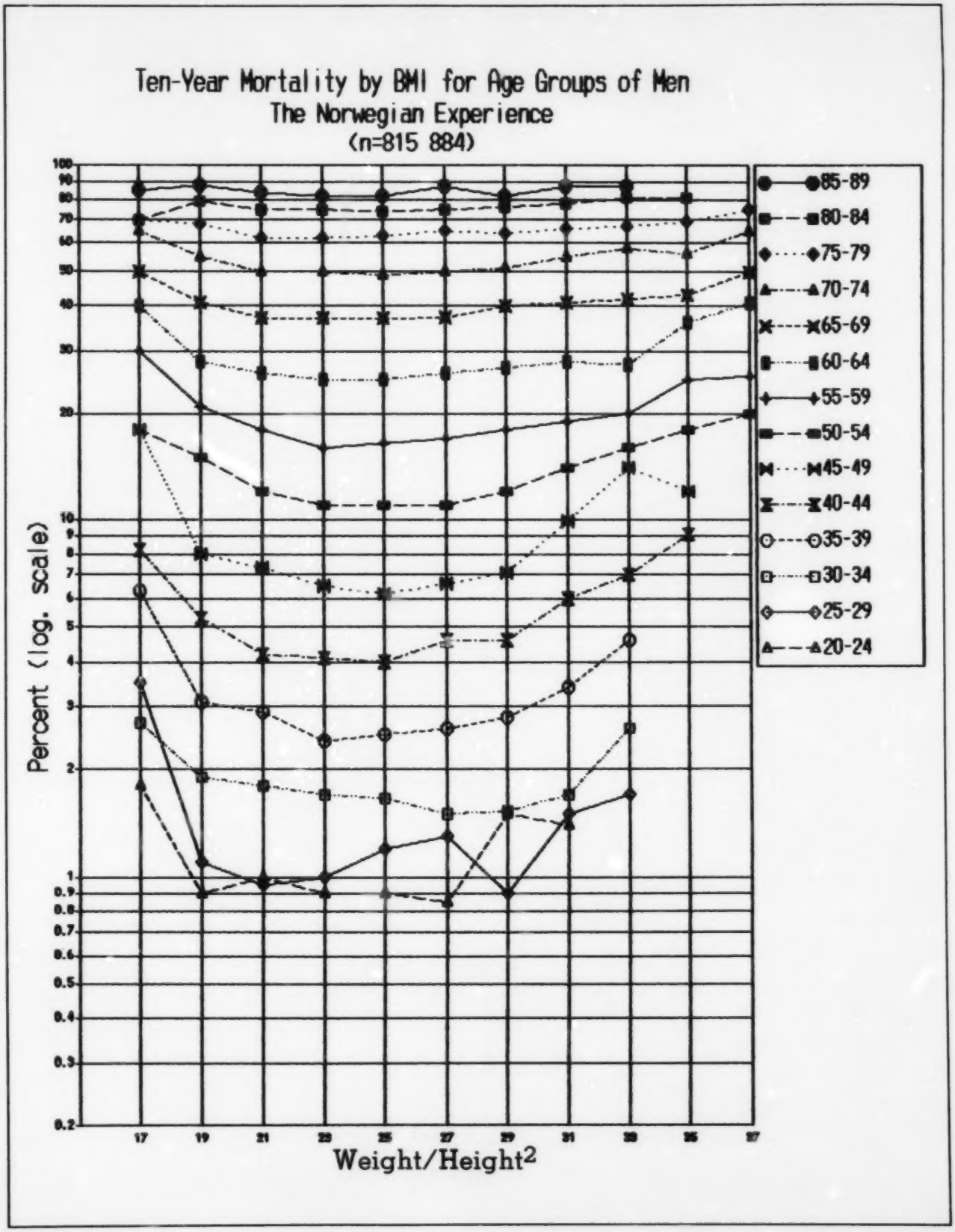

Ten-Year Mortality by BMI for Age Groups of Men
The Norwegian Experience
(n=815 884)
Percent (log. scale)
Weight/Height2
85-89
80-84
75-79
70-74
65-69
60-64
55-59
50-54
45-49
40-44
35-39
30-34
25-29
20-24

Figure 2.

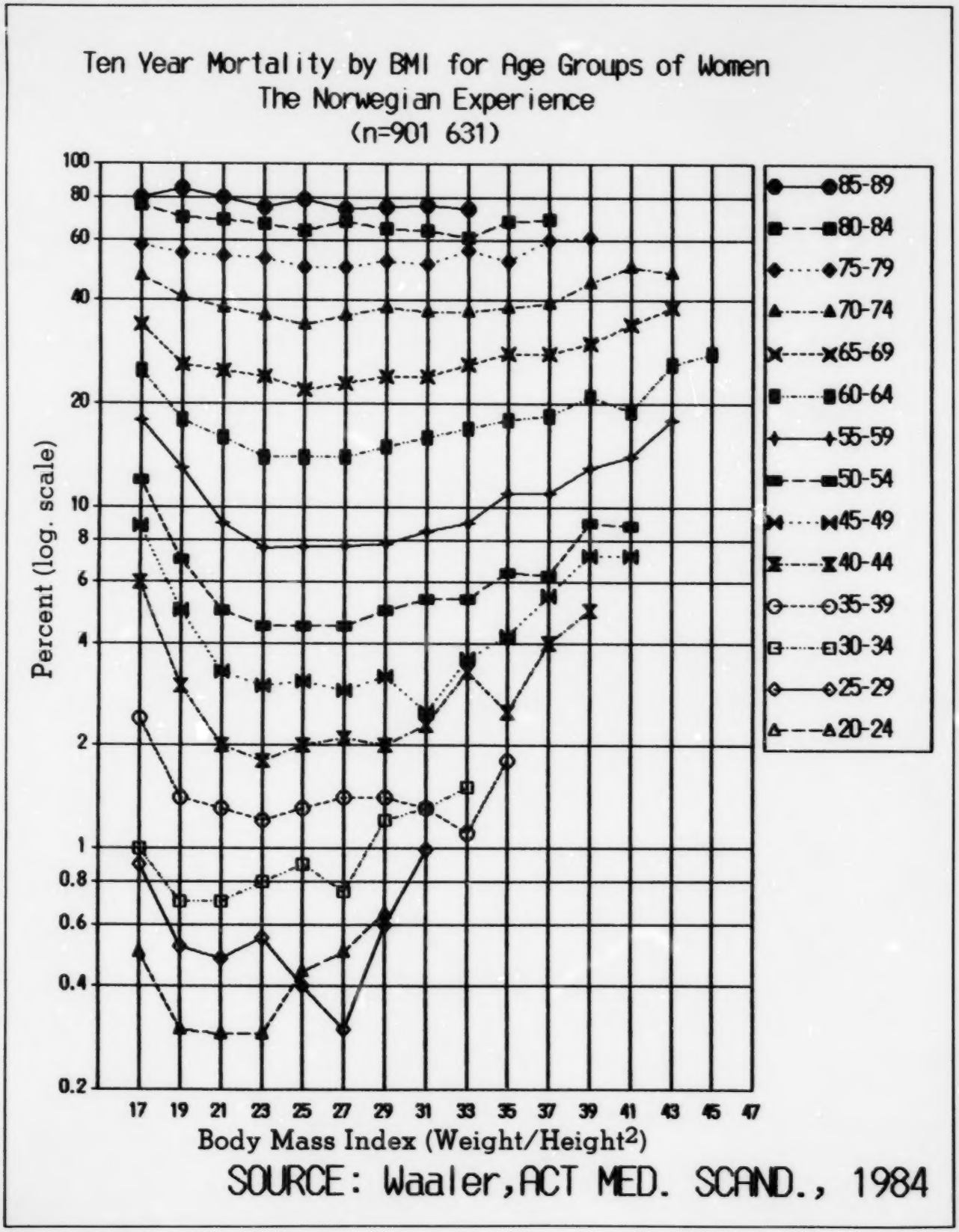

Ten Year Mortality by BMI for Age Groups of Women
The Norwegian Experience
(n=901 631)
Percent (log. scale)
100
80
60
40
20
10
8
6
4
2
1
0.8
0.6
0.4
0.2
17 19 21 23 25 27 29 31 33 35 37 39 41 43 45 47
Body Mass Index (Weight/Height2)
85-89
80-84
75-79
70-74
65-69
60-64
55-59
50-54
45-49
40-44
35-39
30-34
25-29
20-24
SOURCE: Waaler, ACT MED. SCAND., 1984

Figure 3.

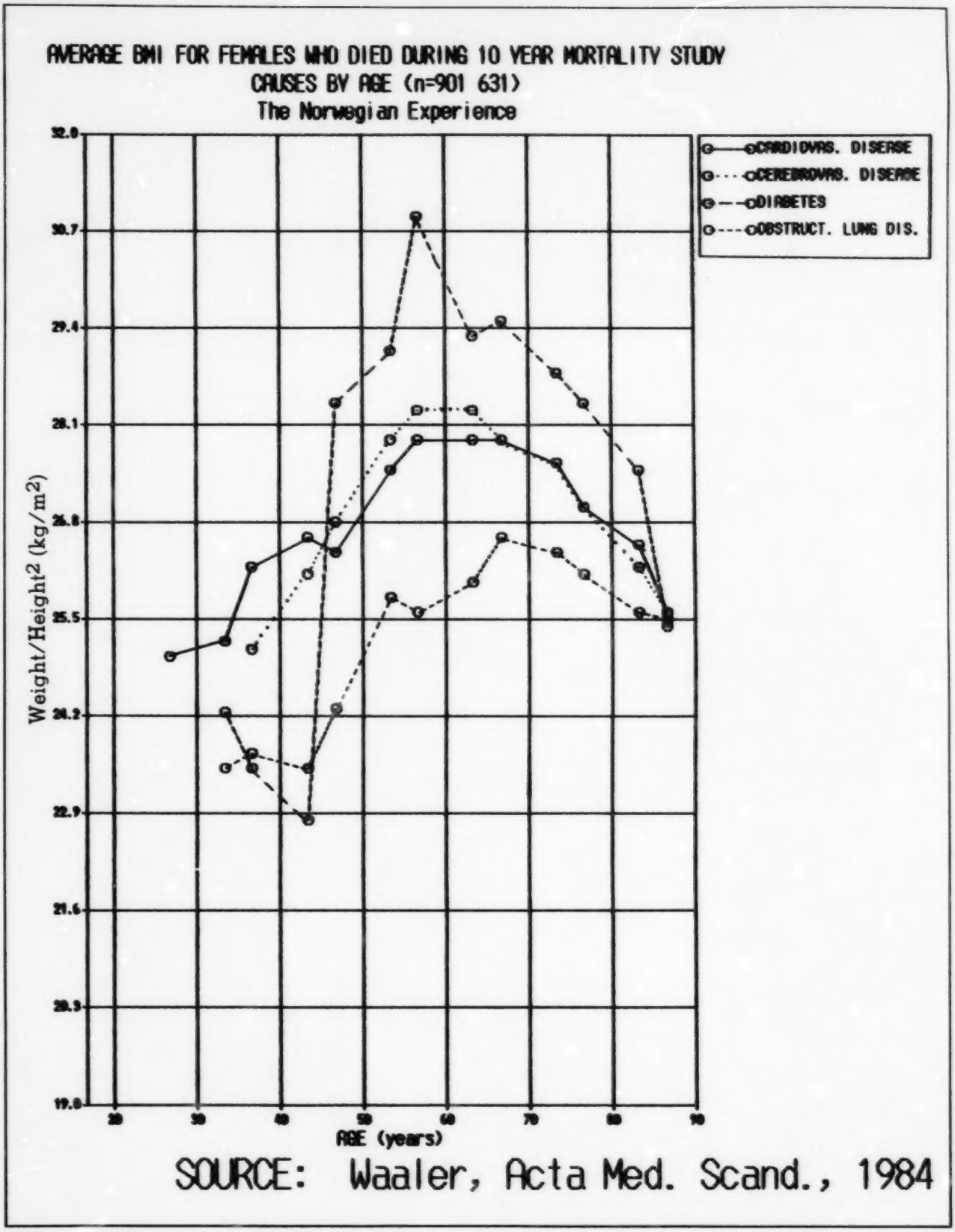

AVERAGE BMI FOR FEMALES WHO DIED DURING 10 YEAR MORTALITY STUDY
CAUSES BY AGE (n=901 631)
The Norwegian Experience
CARDIOVAS. DISEASE
CEREBROVAS. DISEASE
DIABETES
OBSTRUCT. LUNG DIS.
Weight/Height² (kg/m²)
32.0
30.7
29.4
28.1
26.8
25.5
24.2
22.9
21.6
20.3
19.0
20
30
40
50
60
70
80
90
AGE (years)
SOURCE: Waaler, Acta Med. Scand., 1984

Figure 4.

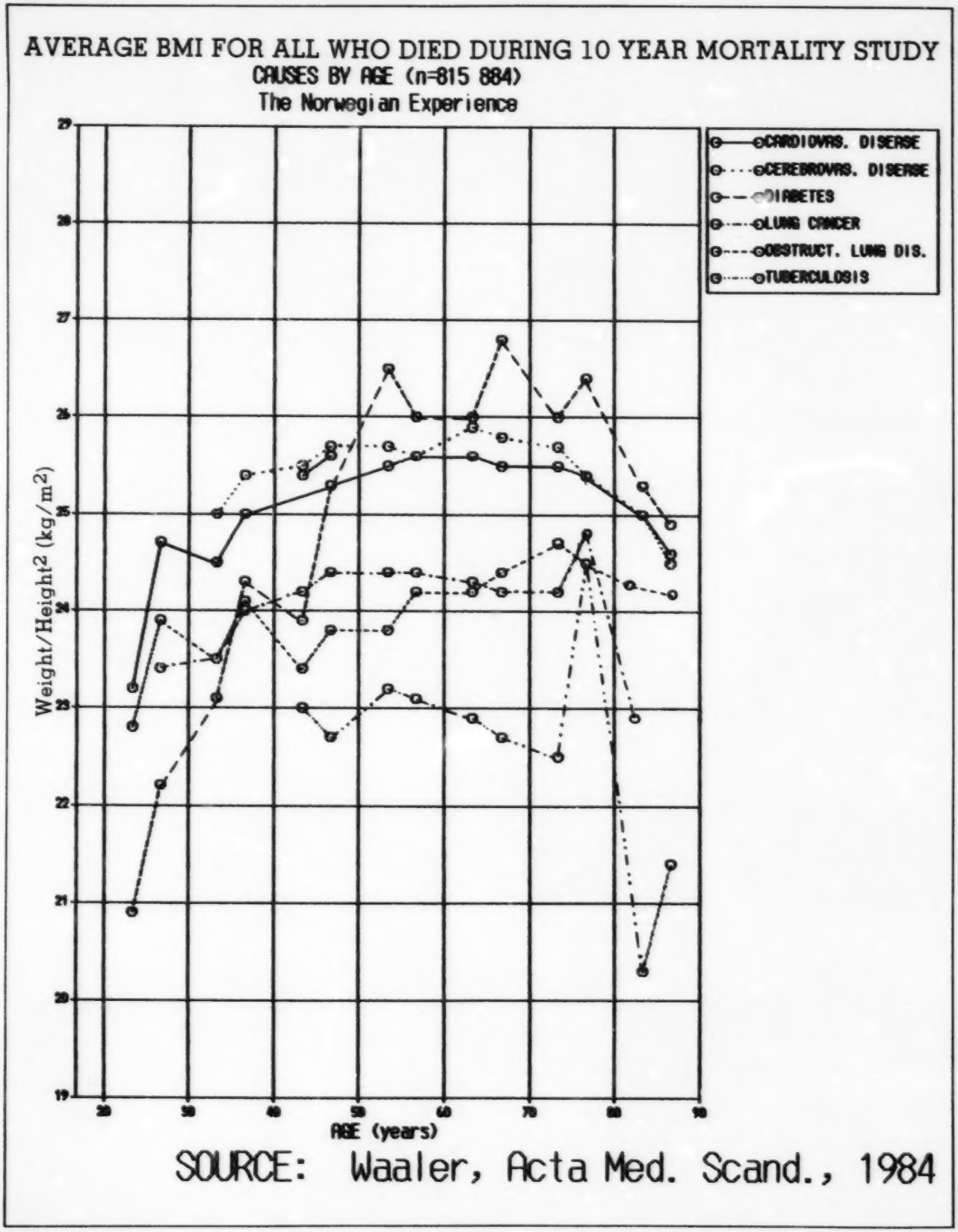
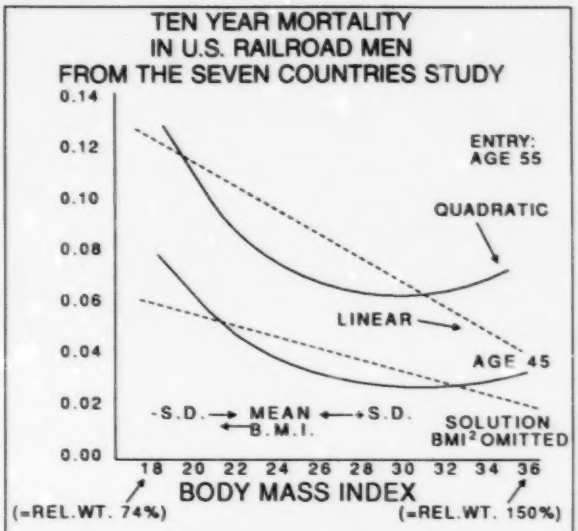

AVERAGE BMI FOR ALL WHO DIED DURING 10 YEAR MORTALITY STUDY
CAUSES BY AGE (n=815 884)
The Norwegian Experience
CARDIOVRS. DISERSE
CEREBROVRS. DISERSE
DIABETES
LUNG CANCER
OBSTRUCT. LUNG DIS.
TUBERCULOSIS
Weight/Height2 (kg/m2)
AGE (years)
SOURCE: Waaler, Acta Med. Scand., 1984

Figure 5.



Source: Keys, Seven Countries, 1980

TABLE 1

14-YEAR MORTALITY BY THE BODY MASS INDEX IN MEN AGED 40-59 (Chicago Peoples Gas Co. Study)

Group	BMI	No.	Rate per/1000 all causes	CVR* diseases	CHD**
ALL	≤23.5	243	288	128	95
(n=1233)	23.6-25.6	254	189	83	63
	25.7-27.3	263	194	122	76
	27.4-29.2	228	127	66	48
	≥29.3	246	196	106	73
SMOKERS	≤23.5	189	312	138	111
	23.6-25.6	160	219	94	69
	25.7-27.3	138	254	159	101
	27.4-29.2	116	147	78	60
	≥29.3	109	229	101	83
NON-SMOKERS	≤23.5	54	204	93	37
	23.6-25.6	94	138	64	53
	25.7-27.3	125	128	80	48
	27.4-29.2	112	107	54	36
	≥29.3	136	169	110	66

SOURCE: Dyer et al., J. Chron. Dis., 1975
*CVR - cardiovascular renal disease
**CHD - coronary heart disease

Figure 6.

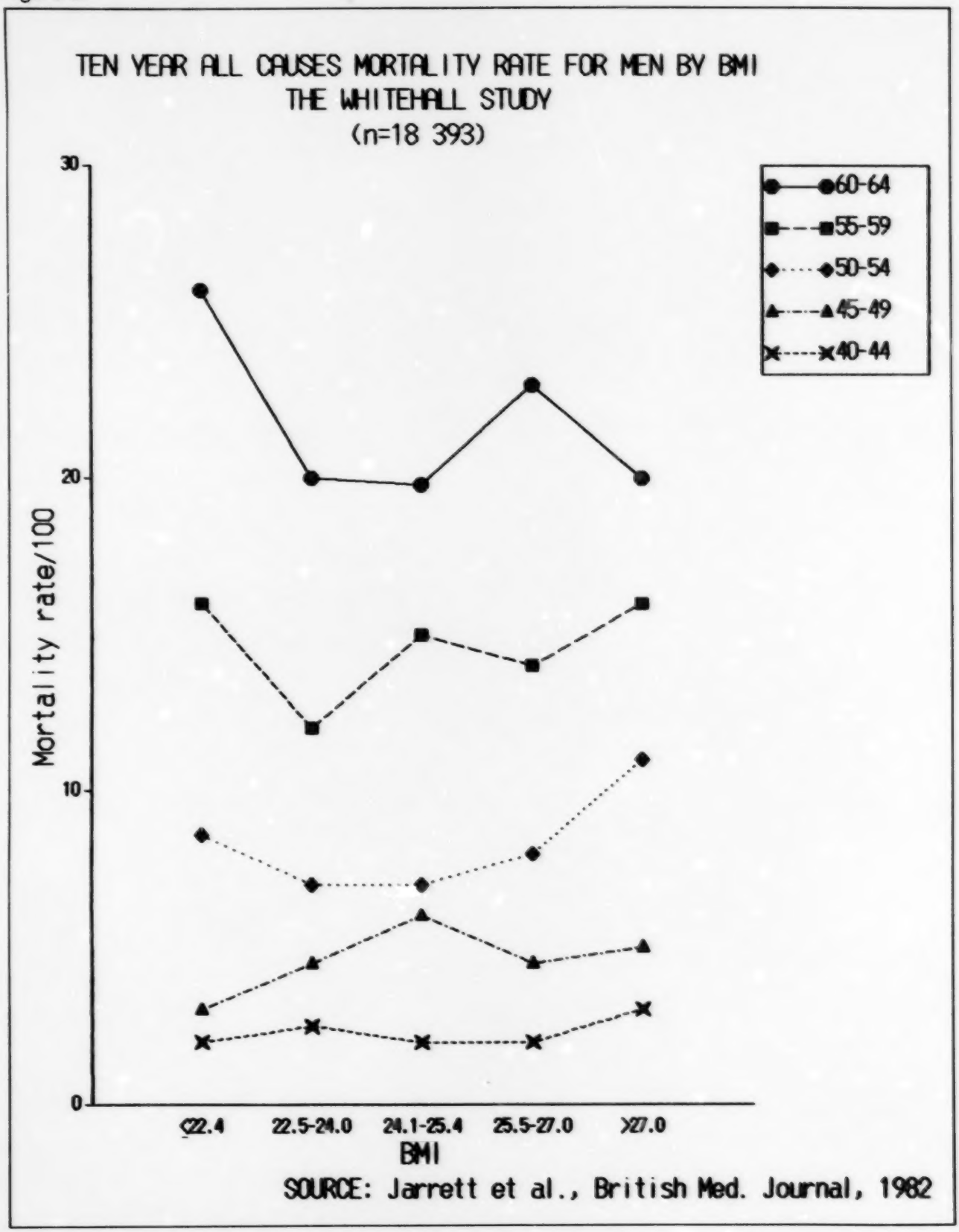

TEN YEAR ALL CAUSES MORTALITY RATE FOR MEN BY BMI
THE WHITEHALL STUDY
(n=18 393)
60-64
55-59
50-54
45-49
40-44
30
20
10
0
Mortality rate/100
<22.4
22.5-24.0
24.1-25.4
25.5-27.0
>27.0
BMI
SOURCE: Jarrett et al., British Med. Journal, 1982

TABLE 2

TEN-YEAR CORONARY HEART DISEASE MORTALITY IN MEN BY THE BMI IN THE WHITEHALL STUDY(n=18 393)

(NO. OF DEATHS (*MORTALITY RATIO*))

BMI	AGE 40-49	50-59	60-69	All Ages	n
≤22.4	20 (1.00)	70 (1.00)	39 (1.00)	129 (1.00)	3846
22.5-24.0	24 (1.32)	66 (0.94)	31 (0.96)	121 (1.00)	3607
24.1-25.4	27 (1.48)	78 (1.07)	39 (1.02)	144 (1.12)	3745
25.5-27.0	24 (1.49)	77 (1.12)	45 (1.11)	146 (1.16)	3494
27+	24 (1.55)	116 (1.51)	47 (1.01)	187 (1.36)	3701

SOURCE: Jarrett et al., British Medical Journal, 1982

TABLE 3

AGE-ADJUSTED SIX-YEAR MORTALITY AND THE BMI

The Framingham Study

BMI*	MEN (n=5 146)	WOMEN (n=6 829)
< 21	8.8	3.7
21-25	5.4	2.6
26-28	4.5	2.7
29-31	5.7	2.7
32-38	3.9	4.1

*Calculated from height/weight table used to define Build Group 1-5.

SOURCE: Sorlie et al., JAMA, 1980.

TABLE 4

RISK OF ISCHEMIC HEART DISEASE AND ITS MANIFESTATIONS BY THE BMI IN 3 983 MEN

after 26 years of observation
The Manitoba Study

Ischemic heart disease (n)	MORBIDITY RATIO (observed over expected) BMI <22.6	22.6-25.0	25.1-27.5	>27.5	P-value
All ischemic heart disease (390)	.66	1.07	1.21	1.28	.01
Sudden Death (S.D.) (177)	.55	.95	1.42	1.85	.01
S.D. without M.I. (82)	.40	1.10	1.32	1.67	.01
Myocardial Infarction (M.I.) (209)	.68	1.08	1.12	1.35	.05
M.I. without S.D (174)	.74	1.18	1.01	1.16	N.S.
M.I. with S.D. (35)	.39	.61	1.63	2.24	.05
Coronary insufficiency or suspected M.I. (131)	.52	.89	1.53	1.46	.05
Angina Pectoris (50)	.92	1.15	1.06	.61	N.S.

SOURCE: Rabkin et al., Am. J. Cardiology, 1977

TABLE 5

AGE-ADJUSTED NINE-YEAR MORTALITY RATES BY DIASTOLIC BLOOD PRESSURE AND THE BMI

(1727 men aged 50-79)

		All causes		Cardiovascular disease		Ischemic heart disease	
		BMI <27	BMI ≥ 27	BMI <27	BMI ≥ 27	BMI <27	BMI ≥ 27
No exclusions (n=1727)							
rate/100	DBP <95 mm Hg	22.0	21.9	11.2	10.0	8.1	8.0
rate/100	DBP ≥95 mm Hg	32.2	21.5	17.9	13.9	12.4	6.8
Relative risk		1.5[B]	1.0	1.6[C]	1.4	1.5	0.9
Excluding all with a history of heart failure or heart disease (n=1372)							
rate/100	DBP <95 mm Hg	19.8	22.0	9.0	9.0	6.1	9.9
rate/100	DBP ≥95 mm Hg	31.8	21.9	15.5	12.6	9.1	5.7
Relative risk		1.6[B]	1.0	1.7[A]	1.4	1.5	0.6
Excluding all who died within 2 yr of follow-up (n=1649)							
rate/100	DBP <95 mm Hg	18.4	18.6	8.6	7.7	5.9	6.3
rate/100	DBP ≥95 mm Hg	26.1	20.2	13.5	12.5	8.7	6.0
Relative risk		1.4[A]	1.1	1.6[A]	1.6	1.5	1.0
Excluding all with a history of diabetes (n=1605)							
rate/100	DBP <95 mm Hg	21.7	28.1	11.2	9.8	8.0	7.7
rate/100	DBP ≥95 mm Hg	34.8	21.5	18.9	13.3	13.1	5.7
Relative risk		1.6[B]	0.8	1.5[B]	1.4	1.6[A]	0.7
Excluding all current smokers (n=1390)							
rate/100	DBP <95 mm Hg	20.3	22.2	10.6	9.7	8.1	7.6
rate/100	DBP ≥95 mm Hg	31.5	20.1	17.3	12.1	11.2	6.1
Relative risk		1.6[A]	0.9	1.6	1.2	1.4	0.3
Excluding all on antihypertensive medication (n= 1447)							
rate/100	DBP <95 mm Hg	20.2	22.3	9.4	9.3	6.7	7.6
rate/100	DBP ≥95 mm Hg	25.6	18.0	14.5	11.1	9.9	5.7
Relative risk		1.3[B]	8.0	1.5	1.2	1.5	0.8

BMI = body mass index (kg/m²): DBP = diastolic blood pressure
[A]p < .05: [B]p <.01: [C]p <.001.
SOURCE: Barrett-Connor and Khaw, Circulation, 1985

TABLE 6

AVERAGE CHOLESTEROL LEVELS BY BMI AND AGE

	BMI							
	MEN (n=1224)				WOMEN (n=1212)			
AGE	<22.5	22.5-25.5	25.5-28.5	>28.5	<21.5	21.5-24.5	24.5-27.5	>27.5
20-29	180	199	202	219	188	199	193	200
30-39	198	204	216	230	200	198	206	209
40-49	214	217	227	249	208	212	225	232
50-59	220	237	237	215	239	250	265	240
60-69	258	223	234	227	256	245	270	242

SOURCE: Simpson et al., Int. J. Epidemiology, 1982

TABLE 7

AVERAGE LEVELS OF PLASMA LIPIDS AND LIPOPROTEINS IN LOWEST AND HIGHEST QUINTILE OF BMI IN 107 HEALTHY MEN

Olivetti Survey - Italy

	BMI (approx. ≤23)	BMI (approx. ≥28)	P-value
Cholesterol (mg/dl)	170	212	<.01
HDL	51	46	
LDL	105	117	
VLDL	13	35	<.001
Triglyceride (mg/dl)	74	165	<.001
HDL	9	10	
LDL	17	21	
VLDL	43	127	<.001

HDL = High density lipoprotein
LDL = Low density lipoprotein
VLDL = Very low density lipoprotein

SOURCE: Fainaru et al., Medical Complications of Obesity, 1979

TABLE 8

PREVALENCE (%) OF ADULT ONSET DIABETES: U.S., 1976

(n=64 142)

	BMI (W/HT)							
	MEN				WOMEN			
AGE	<24	24-25	26-27	28+	<22	22-25	26-27	28+
20-44	0.5	0.5	0.6	1.3	0.5	0.6	1.1	3.4
45-54	3.0	2.5	3.4	6.9	1.8	2.1	3.6	9.1
55-64	4.3	4.7	5.9	9.9	2.5	3.7	5.1	9.6
65+	6.7	8.0	8.2	9.4	5.6	6.4	8.9	13.2

SOURCE: Bonham and Brenk, Am. J. Clin. Nutr., 1985

TABLE 9

FIVE-YEAR INCIDENCE OF DIABETES IN MEN AGED 40 AND OVER BY BMI

Israeli Study (n=8688)

	Rate/1000			
BMI	AGE 40-49	AGE 50-59	AGE ≥60	ALL
<24.2	15	30	23	23
24.2-27.0	30	47	38	38
>27.0	46	71	58	58

SOURCE: Medalie et al., Arch. Intern. Med., 1985

TABLE 10

RELATIVE RISK OF BREAST CANCER BY BODY MASS INDEX SAN FRANCISCO

(case-control study)

BMI	Premenopausal (n=1098)	Postmenopausal (n=2807)
<21.5	1.00	1.00
21.5-24.4	.91	1.28
≥24.5	.65	1.39

SOURCE: Paffenbarger, Amer. J. Epidemiology, 1980

TABLE 11

BREAST CANCER AND THE BMI IN WOMEN AGED 60 AND OVER

(case-control study, cases-431, controls-372)

Israel

BMI	Crude odds ratio	Adjusted odds ratio*
≤19	1.00	1.00
19.1-23	2.16	1.78
23.1-27	2.44	1.92
>27	2.99	2.53
	P<.001	P<.01

* adjusted for age at menarche, first birth and menopause, years of education, number of births, history of benign breast disease, family history of breast cancer.

SOURCE: Lubin et al., Amer. J. Epidemiology, 1985

TABLE 12

RELATIVE RISK OF BREAST CANCER BY BMI POLAND

(case-control study, n=1443)

BMI	AGE 25-49	AGE 50-69
<24.0	1.00	1.00
24.0-26.9	.81	1.59
27.0-29.9	.73	1.90
30.0+	.99	2.08

SOURCE: Staszewski, Preventive Medicine, 1977

TABLE 13

DISTRIBUTION OF 173 CASES OF ENDOMETRIAL CANCER AND 347 CONTROLS BY BMI AND ESTROGEN USE

	W/H² Index <20		20-24		25-29		30-34		≥35	
	No.	(%)	No.	(%)	No.	(%)	No.	(%)	No.	(%)
Total										
Cases	16	(9)	56	(32)	50	(29)	31	(18)	20	(12)
Controls	61	(18)	164	(47)	95	(27)	23	(7)	4	(1)
Rate ratio	(1)*		1.3		2.0		5.1		19.1**	
(95% CI)			(0.7-2.4)		(1.0-3.8)		(2.4-10.9)		(6.7-53.9)	
Estrogen non-users										
Cases	9	(8)	36	(30)	30	(25)	26	(22)	19	(16)
Controls	56	(19)	135	(46)	80	(27)	18	(6)	3	(1)
Rate ratio	(1)*		1.7		2.3		9.0		39.4***	
(95% CI)			(0.8-3.7)		(1.0-5.2)		(3.8-21.5)		(12.4-124.6)	
Estrogen users										
Cases	7	(13)	20	(38)	20	(38)	5	(9)	1	(2)
Controls	5	(9)	29	(52)	15	(27)	5	(9)	1	(2)
Rate ratio	(1)*						1.6			
(95% CI)							(0.7-3.4)			

* Reference category
** Test for trend: $X^2_1 = 45.3$; $p<0.001$
*** Test for trend: $X^2_1 = 55.4$; $p<0.001$

SOURCE: La Vecchia et al., Int. J. Epidemiology, 1982

TABLE 14

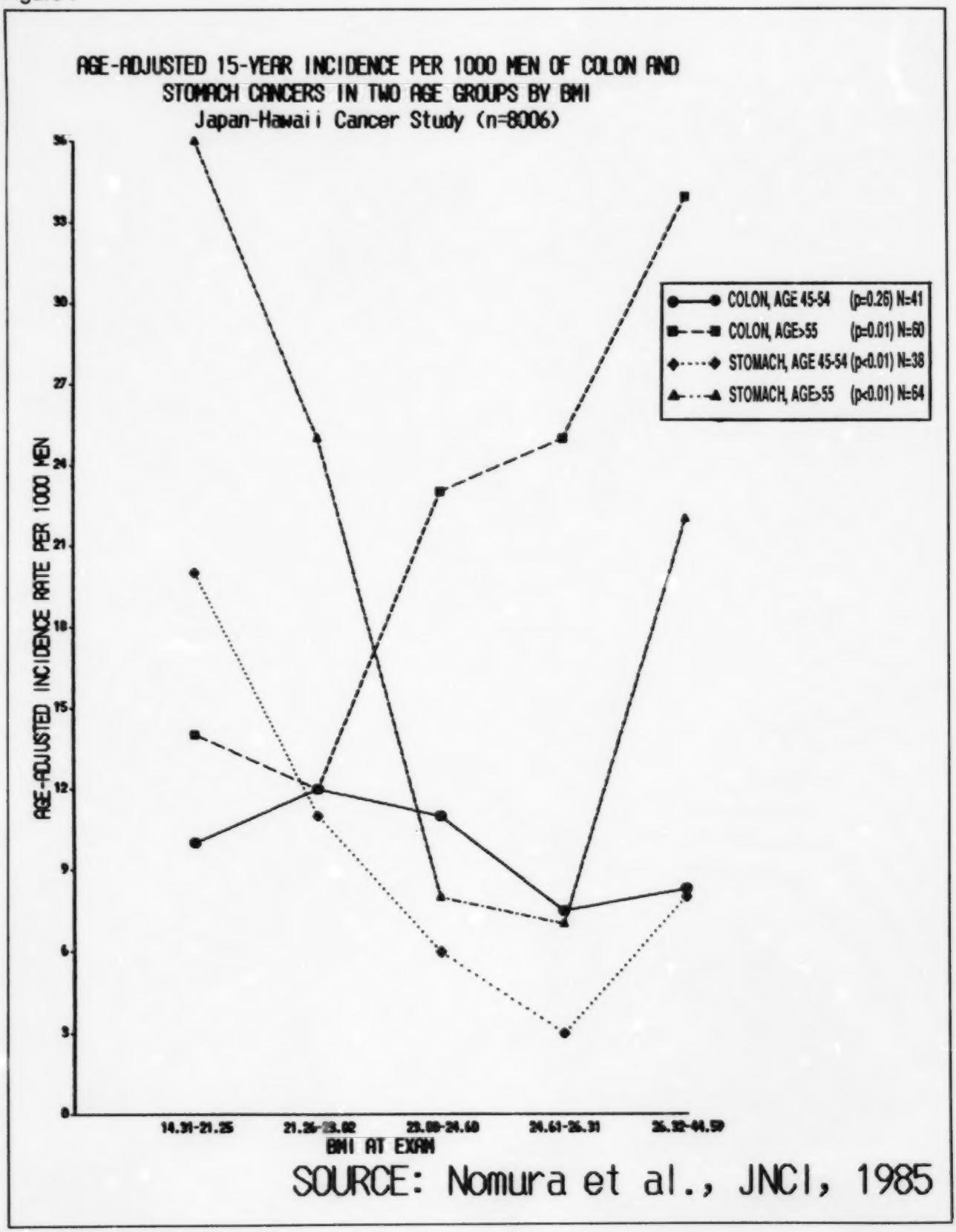
15-YEAR AGE-ADJUSTED CANCER INCIDENCE PER 1 000 MEN BY BMI (n=8006)

Japan - Hawaii Cancer Study

BMI	Stomach n=104	Colon n=101	Prostate n=96	Rectum n=63	Lung n=101
14.3-21.3	26.5	12.0	11.4	6.4	16.8
21.3-23.0	15.8	11.6	9.8	11.8	12.0
23.0-24.6	10.3	15.7	15.4	10.9	12.3
24.6-26.3	4.2	12.8	13.3	6.2	14.9
26.3-44.6	12.3	17.4	14.2	5.8	11.6
	p<.001	p=.167	p=.241	p=.294	p=.383

SOURCE: Nomura et al., JNCI, 1985

Figure 7


AGE-ADJUSTED 15-YEAR INCIDENCE PER 1000 MEN OF COLON AND STOMACH CANCERS IN TWO AGE GROUPS BY BMI
Japan-Hawaii Cancer Study (n=8006)
AGE-ADJUSTED INCIDENCE RATE PER 1000 MEN
COLON, AGE 45-54 (p=0.26) N=41
COLON, AGE>55 (p=0.01) N=60
STOMACH, AGE 45-54 (p<0.01) N=38
STOMACH, AGE>55 (p<0.01) N=64
14.31-21.25
21.26-23.02
23.03-24.60
24.61-26.31
26.32-44.59
BMI AT EXAM
SOURCE: Nomura et al., JNCI, 1985

APPENDIX III

REPORT ON CLINICAL-METABOLIC EVIDENCE FOR THE IMPACT OF OBESITY ON CARDIOVASCULAR RISK FACTORS

Prepared by:
Dr. Daniel A.K. Roncari

Prepared under contract with the Department of National Health and Welfare

Appendix III

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	(a) Impact of Obesity on Cardiovascular Risk Factors	114
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I. INTRODUCTION

Since publication (1983) and submission of the 1986 update of my relatively extensive review on OBESITY,[1] the full report of the National Institutes of Health Consensus Development Conference on HEALTH IMPLICATIONS OF OBESITY, held on 11-13 February 1985 was published as Part 2 of the December 1985 issue of the Annals of Internal Medicine.[2]

While the Consensus Conference Statement[3] and some other associated papers[4-6] emphasize rather strongly the numerous harmful consequences of obesity, including those on the cardiovascular system, the heterogeneous nature of the "obesity syndrome" was relatively neglected. Indeed, the Consensus Statement and some of the other papers, underscore the "continuous" correlation between body weight or body mass index and complications, including those injurious to the cardiovascular system. More specifically, in the Conclusions of the Consensus Statement, the general statement is made that "treatment is strongly advised" for adult Americans with "a body mass index greater than 27.8 kg/m^2 (men) or 27.3 (women)"[3]. This level of adiposity is quite close to a body weight increase of 20% above desirable. Even lesser degrees of adiposity should apparently be treated in the presence of certain associations and other circumstances.[3]

Such conclusions are somewhat surprising because of statements enunciated by some other papers of this December 1985 issue. For example, the stronger association between regional distribution of adipose tissue and cardiovascular complications, than with obesity in general, is rather convincing.[7] One paper concludes that obesity (apparently directly or indirectly (?)) does not cause atherosclerosis,[8] and some papers also imply that studies of insured groups and prospective epidemiological investigations, e.g. the Framingham Study, may miss special groups, including those with massive corpulence (more than 170-200% of desirable levels), or those with particular adipose configuration, or those in lower socio-economic strata (who generally have higher morbidity and mortality, and fewer of whom are insured).[1,9] Papers also emphasize the decreasing impact of obesity, already after 45 years of age,[1,4,10,11] and the need to re-adjust levels of "desirable" weight "for older adults".[10]

In this report, section (a) describes some background knowledge associating the impact of obesity on cardiovascular risk factors.[1] Then, the discussion becomes more specific: (b) describes possible evidence in favour of an independent influence of obesity on atherogenesis. Section (c) discusses the differential effects of obesity as pertinent to adipose distribution (other differential effects were already dealt with under (a), subsection on diabetes). Before the "Concluding Remarks", section (d) restates the diminishing influence of obesity in relation to age. The Concluding Remarks are somewhat at variance with the "standard" literature on obesity, and should provoke animated discussion.

(a) Impact of Obesity on Cardiovascular Risk Factors

As stated and as will be specified later, in this writer's opinion, it is impossible to describe the precise extent to which obesity imparts its nociferous influence on cardiovascular risk factors in a heterogeneous population of obese persons. In any case, this section will present the harmful associations in generic terms.

The Framingham Study, a monumental series of epidemiologic investigations, has made it clear that the effects of obesity on the cardiovascular system are mediated in great part by the following common pathophysiologic associations: hypertension, decreased levels of high-density lipoprotein-cholesterol (HDL), hypercholesterolemia due to elevated concentrations of low-density lipoproteins (LDL), and diabetes mellitus.[5,11,12,13]

A description of the pathophysiological mechanisms whereby the various metabolic abnormalities may lead to cardiovascula-complications follows.

Glucose intolerance. Obesity is commonly associated with impaired glucose tolerance.[14,15] The probability of developing non-insulin-dependent (Type II) diabetes is positively correlated with the duration (and to some extent the degree) of adiposity, but in a given patient the time of onset and severity of glucose intolerance depend on a variety of interacting genetic and environmental factors.[14,15] The impaired glucose tolerance associated with obesity is characterized by basal and stimulated (e.g. post-prandial) hyperinsulinemia.[16,17,18] Many obese persons have relatively mild glucose intolerance, only detectable by a glucose tolerance test or a plasma glucose determination, 2 hours following the administration of a standardized quantity of carbohydrate. Some obese subjects have relatively pronounced impairment, including fasting hyperglycemia, i.e. Type II diabetes mellitus. Although still hyperinsulinemic, they have relatively lower insulin levels than obese persons who have retained satisfactory glucose tolerance.[19] Only a small fraction of obese persons with glucose intolerance develop insulin-dependent (Type I) diabetes; such unusual superimposition of one form of diabetes on another is heralded by otherwise unaccounted, substantial weight loss, with increasing polyuria and polydipsia.

The concurrence of hyperglycemia and hyperinsulinemia reflects the resistance of target cells to insulin, characteristic of obesity.[14,15,20-24] Target cells are more resistant to certain insulin action, for example, stimulation of glucose transport compared to inhibition of lipolysis. Although fat cells share the insulin resistance of other cells, adipose tissue accounts for only a small fraction of the total carbohydrate metabolized by the body.[25] In fact, marked improvement in the hyperinsulinemia and insulin insensitivity frequently becomes evident after only modest loss of body fat. Consequently, overeating and physical inactivity, which contribute to the development of hyperinsulinemia, may be crucial factors in conferring resistance to insulin. The impact of target-cell resistance to impaired glucose tolerance is mediated mainly by insensitivity of skeletal muscle and liver. The resistance of myocytes is principally responsible for decreased glucose utilization, while that of hepatocytes (complemented by exaggerated responses to glucagon) for excessive glucogenesis. More recent investigation indicates that, in addition to target cell resistance to insulin, diminished hepatic extraction of insulin contributes appreciably to the hyperinsulinemia of obesity.[26,27] Through its trophic effects (which may be mediated through the insulin-like growth factor I receptor), it has been suggested that elevated insulin levels may promote atherosclerosis by stimulating the proliferation of smooth muscle and other vascular cells; acting through its own receptors, hyperinsulinemia might also exaggerate lipid synthesis in these cells.[28]

The insulin resistance in subjects with mild glucose intolerance is due in great part to a decreased number of insulin receptors. (Even after allowing for the fact that only a small fraction of receptors must be occupied for maximal insulin action).[21-23,29] The diminution in receptor number is secondary to several factors including hyperinsulinemia, excessive nutrient energy, high-carbohydrate and high-fat diets, and physical inactivity.[21,22] When fasting hyperglycemia develops, the insulin resistance results from both a decrement in receptor number and from impairment of post-receptor mechanisms; their dysfunction assumes increasing pathogenetic significance in more severe Type II diabetes.[23,24,27] Post-receptor defects include impaired recruitment of glucose transporters from the cell interior to the surface.[24,30]

For greatest stimulation of glucose transport, 5% to 10% of the insulin receptors must be occupied. By comparison, 1% to 2% of the receptors must be

occupied for maximal inhibition of lipolysis in adipocytes. The lesser requirement, along with selectively lower degrees of insulin resistance as regards the antilipolytic effect[31] (coupled with excessive triglyceride synthesis), explains the progressive accumulation of fat in adipocytes - that is, the aggravation of adiposity under conditions of energy excess, despite resistance to certain effects of insulin. Persistent partial control of lipolysis explains to some extent the rarity of ketoacidosis in diabetes associated with obesity. A related factor is the raised hepatic insulin:glucagon ratio, which leads to relatively high malonyl-CoA levels.[32] This compound is not only elongated to fatty acids at rapid rates (in obesity), but also inhibits the enzyme carnitine acyltransferase I.[32] Consequently, long-chain fatty acids cannot penetrate mitochondria to any appreciable extent, and the limitation of ß-oxidation prevents inordinate ketogenesis. Recently, the antilipolytic action of insulin has been examined in more depth. Studies in vivo and in isolated fat cells from obese subjects, have confirmed relative maintenance (only slight decrease at lower insulin levels) of antilipolysis, in the presence of resistance to insulin-stimulated glucose transport.[33,34]

The hyperinsulinemia and insulin resistance associated with obesity are promptly reversed by appropriate therapeutic measures.[14,21,22] Whereas in obesity (fed state) the affinity of the receptor for insulin remains normal, it is enhanced by a fast of 48 to 72 hours, without changes in receptor number.[21] On the other hand, nutrient energy restriction (and exercise) for several weeks corrects the hyperinsulinemia and restores the appropriate complement of normal-affinity receptors.[21] Both solubleand insoluble types of fibre increase the affinity of insulin receptors.[22]

Another mechanism worsens glucose intolerance. In contrast to insulin-dependent diabetes, patients with Type II diabetes have an exaggerated glucagon response to a protein meal and to arginine.[35] In turn, glucagon stimulates insulin secretion and may aggravate the hyperinsulinemia. Recent studies also indicate that, in Type II diabetes, the glucagon-producing A cells do not respond normally to hyperglycemia by suppressing glucagon secretion, worsening the glucose intolerance.[36]

It may be relevant that the response of the intestinal hormone, glucose-dependent insulinotropic polypeptide (gastric inhibitory polypeptide, GIP) to oral glucose, fat, or mixed meal is apparently normal in obese non-diabetic persons, but blunted in obese (but not lean) subjects with Type II diabetes.[37,38] It remains to be established whether this impairment contributes to the relatively impaired secretion of insulin present in obese diabetic patients.

In addition to insulin resistance and exaggerated glucagon responses, decreased beta-cell responsiveness to glucose and to other secretagogues, contributes to the glucose intolerance associated with obesity.[39] Thus, fasting hyperglycemia may occur despite the fact that additional insulin is available for secretion, as revealed by a further rise in plasma insulin level elicited by a meal. The decreased beta-cell responsiveness (sensitivity) is improved or corrected by therapeutic measures leading to appropriate loss of excessive body fat.[39] Such treatment, moreover, results in restoration of beta-cell capacity; this term signifies that appropriate insulin secretion may be maintained for several months despite resumption of hyperphagia and recurrence of obesity.[39]

The frequent coexistence of diabetes mellitus and decreased levels of HDL confers a strong atherogenic influence, particularly in obese women.[13] In fact,

diabetic women have a relatively greater mortality from CHD than diabetic men.[13] This higher death rate contrasts with the appreciably greater vulnerability to coronary heart disease of non-diabetic men relative to women.

Dyslipoproteinemias. Hepatic synthesis of cholesterol and triglyceride, as well as their incorporation into very-low-density lipoproteins (VLDL), are accelerated in obese persons.[39-41,42,43] These and other metabolic abnormalities are particularly prominent during the development and progression of adiposity. Increased provision of substrates, including glucose, amino acids and fatty acids (the latter are formed de novo and derived by augmented mobilization from adipose tissue), contribute to excessive VLDL-triglyceride formation and secretion. Furthermore, hyperinsulinemia associated with obesity may contribute to the stimulation of this synthetic process, as reflected by the frequent hypertriglyceridemia.[44,45]

Despite excessive hepatic VLDL production, many obese persons have normal plasma triglyceride concentrations because their plasma clearance is increased.[41] Enhanced synthesis in and release from adipocytes of lipoprotein lipase, are at least partly responsible for the promotion of triglyceride clearance.[41] On the other hand, the obese state triggers or aggravates hypertriglyceridemia in subjects with underlying genetic abnormalities characterized by VLDL-triglyceride (and VLDL-remnant-triglyceride) overproduction and/or clearance (multiple lipoprotein-type hyperlipidemia and primary Type III, IV, and V hyperlipoproteinemias). It remains to be definitely established whether elevated VLDL-triglyceride levels confer an independent atherogenic influence (increased chylomicron-triglycerides are not atherogenic) when allowance is made for the contribution of such frequently coexistent atherogenic factors as hypercholesterolemia, decreased HDL and hypertension.[12,13,46]

On the capillary endothelial surface, lipoprotein lipase cleaves VLDL-triglycerides to form intermediate-density lipoproteins (VLDL-remnants). Their triglycerides are in turn hydrolyzed to produce LDL, the major carriers of cholesterol to tissues. Augmentation of these conversions contributes to the modest positive correlation between the level of plasma cholesterol, specifically LDL-cholesterol, and obesity. Since assimilation of fatty acids from circulating VLDL-triglycerides into adipocyte triglycerides is also stimulated, and in view of excessive mobilization of fatty acids from adipose tissue, in turn resulting in greater hepatic VLDL-triglyceride synthesis, the entire "adipo-hepatic cycle" is accelerated.[41,47] Consequently, the traffic of the atherogenic LDL and intermediate-density lipoproteins proceeds at an accelerated pace. Indeed, recent investigations have revealed that both LDL and apolipoprotein B production and turnover are accelerated in obesity and in mild Type II diabetes (in more severe non-insulin-dependent diabetes, removal of LDL is also delayed).[49,50] The augmented flux and load of LDL, which frequently occur despite normal circulating levels, would favour atherosclerosis. Incidentally, the raised concentrations of glycated LDL present in poorly controlled diabetes, like other modified LDL, might be more injurious to the arterial wall than normal LDL molecules.

A strong negative correlation exists between plasma HDL levels and obesity.[12,13] The decrease in HDL complements elevation of LDL (a highly atherogenic combination) in leading to accumulation of cholesterol in extra-hepatic tissues.[42,48] Indeed, the total body cholesterol content is expanded in obese subjects, mostly because of excessive storage, mainly as unesterified cholesterol, in fat cells.[42] The increased

fat tissue cholesterol pool, which is in slow equilibrium with circulating cholesterol, contributes to the exaggerated turnover of this sterol.[51] As a result of these metabolic derangements, excessive amounts of the atherogenic LDL and intermediate-density lipoproteins (when formed excessively, some of these are not converted further) reach the arterial wall and contribute to the disruption of endothelial integrity; the deposited lipoprotein particles, along with their cholesterol and cholesterol esters, participate in the formation of atheromas. These pathologic processes interact synergistically with such other cardiovascular factors as hypertension.

Hypertension. Both systolic hypertension and diastolic hypertension are common cardiovascular risk factors in obesity.[11,52-54] The consequence of this significant association is emphasized by the organization of a Satellite Symposium (entitled Obesity and Hypertension) to the Third International Congress on Obesity. The proceedings of that Symposium were published in supplement 1 of volume 5, International Journal of Obesity, 1981.

Hypertension induced or aggravated by obesity has characteristics distinct from "essential hypertension" in lean subjects. Hypertension associated with obesity is featured by volume overload and actually decreased total peripheral resistance.[55] More specifically, these obese subjects reveal augmented cardiac output, stroke volume, central blood volume, as well as expanded plasma and total blood volume.[55] Should obesity and essential hypertension (typical of the lean) occur together, the effect of adiposity on peripheral vascular resistance would mitigate its increase in essential hypertension, a beneficial effect particularly evident in the renal vasculature. However, the increased (hypervolemic) ventricular preload superimposes on the peripheral resistance of essential hypertension, exacting a "double burden" on the left ventricle, increasing the probability of developing hypertrophy and failure.[55]

The association between obesity and high blood pressure is stronger in women.[53] Hypertension is also more common in adult-onset, moderate (hypertrophic) obesity and, relatedly, in persons with truncal deposition of fat.[56] Furthermore, a notable correlation between hypertension and obesity is also evident in children and adolescents of both sexes, an important consideration for early treatment and prevention.[53] It should be noted that small sphygmomanometer cuffs may give falsely high readings; blood pressure values obtained with appropriately large cuffs reflect accurately direct intraarterial determinations.[57]

The pathogenesis of hypertension in obesity has not been completely elucidated. (Rare endocrine syndromes, such as Cushing's syndrome, which contribute to the development of both obesity and hypertension, are excluded from this discussion.) Genetic factors render some obese persons vulnerable to the development of high blood pressure. Overeating and periods of re-feeding lead to overactivity of the sympathoadrenal system.[58,59] Increased release of catecholamines contributes to the development of hypertension. Norepinephrine also enhances sodium reabsorption in the distal nephron. In addition, overeating increases T_3 relative to reverse T_3 levels, thus promoting the action of catecholamines on target cells.[59,60] Of great import, moreover, increased sympathetic nervous system activity enhances renin secretion.[58,61] Renin leads to increased formation of angiotensin II, which in turn results in exaggerated production of aldosterone, the most powerful promoter of sodium reabsorption by distal renal tubules. A hypothesis to be tested proposes increased production of a peptide derived from pituitary adrenocorticotropin-melanotropin-endorphin precursor, which

leads to augmented aldosterone production in the obese.

Insulin stimulates sodium reabsorption in the distal nephron.[59,62] Such an effect is possible because renal tubules maintain relative sensitivity to insulin.[59] The elevated insulin levels might also contribute to the excessive ß-adrenergic activity by stimulating the ventromedial hypothalamic-sympathetic nervous system.[63-66] Finally, excessive salt intake during overeating worsens hypertension.

Hypertension, of course, contributes to a major extent to the increased incidence of strokes, cardiomegaly and congestive heart failure in obese subjects.[11,53,54] It is also an important risk factor in the development of atherosclerotic coronary heart and renovascular disease.

Weight loss, even when modest and without sodium restriction, results in significant decreases in blood pressure in hypertensive and "normotensive" subjects.[67] During the first 2 weeks of nutrient energy restriction, the appreciable lowering of blood pressure is due partly to natriuresis and decreased plasma volume.[61] This hypotensive effect may be partly due to decreased plasma insulin levels (or decreased insulin: glucagon ratio).[59,62] Depressed sympatho-adrenal activity may also play a role. Plasma renin activity is not changed at this stage. After the first 2 weeks of nutrient energy restriction, the continued but more gradual fall in blood pressure is associated with a significant decrease in plasma renin activity and with a lesser fall in plasma aldosterone concentration.[61] The smaller decrease in aldosterone may reflect rising levels of potassium during weight loss, since potassium ions stimulate aldosterone secretion directly. Attenuated sympathic nervous system activity, further blunted by the characteristic increase in the production of reverse T_3 relative to T_3 during nutrient energy deprivation, contributes appreciably to the lowered plasma renin activity and decline in blood pressure.

Although nutrient energy restriction and weight loss have significant independent hypotensive effects, moderate salt restriction is still recommended. Regular exercise at levels adequate to achieve physical fitness decreases blood pressure independently of weight loss, even in massively obese patients who have an excessive number of fat cells.[63] Such effect of exercise may be mediated in part by the decreased insulin levels. With appropriate nutrient energy restriction and physical training, hypotensive medications can frequently be stopped or their dose decreased. Should hypertension persist, however, effective pharmacologic therapy should be provided, and any specific corrective cause should be sought.

More recent reports corroborate the beneficial influence of effective therapy of obesity on its associated hypertension Salt restriction does effect a lowering influence, additional to that of weight loss (or antihypertensive mechanisms associated with a successful weight-reducing program), particularly during early treatment.[69,70] Quite recently, the effects of nutrient energy restriction to induce weight loss were compared to those of the beta-adrenergic blocker, metoprolol, in a group of obese, mildly hypertensive subjects.[71] Not only was there a greater decrement in blood pressure in those treated by diet, but their lipoprotein profiles also improved, while those treated with the beta-blocker worsened. In the latter, the ratio LDL-cholesterol/HDL-C increased significantly (similar adverse effects are observed with thiazides), while it decreased significantly in the group that lost weight.[71]

While the limitations that preclude quantification of the influence of obesity on cardiovascular risk factors are discussed in this Report, as generaliza-

tions, and examples, some published data will now be presented. In the relatively selected (whole) population of the Framingham Study, over a relative body weight scale ranging from about -12 to +9 to +12 (0 being the "desirable" value), in men, the approximate relative total serum cholesterol increased from -11 to +9, systolic blood pressure from -3 to +7 and blood glucose from -2 to +2.[72] In women, corresponding values were as follows: total serum cholesterol rose from -4 to +9, systolic blood pressure from -4 to +5 and blood glucose from -2 to +1.[72] As an example of expressing this information in absolute terms, for both men and women, each standard deviation in weight was associated with a 6.5 mm change in systolic blood pressure (i.e. in both directions).[72] Similarly, but expressed differently, other publications related 15% weight gain to an 18% rise in systolic pressure, in men.[73]

As an overall statement, the Framingham Study has indicated that maintenance of "desirable" weight would be associated with a 25% decrease in coronary heart disease, and a 35% reduction in cardiac failure and strokes.[72] Other information on the impact of obesity on cardiovascular risk factors will be presented in the following sections, in which more specific situations or groups are described.

(b) Is Obesity an Independent Atherogenic Risk Factor?

Prolonged follow-up of subjects participating in the Framingham Study has suggested that obesity may be an independent cardiovascular risk factor, i.e., operating through mechanisms additional to those mediating the effects of the known risk factors described in the preceding sections.[74,75] More specifically, and especially in younger participants, increasing degrees of obesity had a progressively greater positive effect on the incidence of coronary heart disease and congestive heart failure in men. In women, increasing corpulence appeared to be an independent risk for coronary heart disease, congestive heart failure and stroke.[75] In addition, weight gain after the early phase of adulthood imposed a further risk for cardiovascular disease, again apparently independently.

It is possible that the additional (over known risk factors) hazard of obesity may be mediated, at least partly, by such characteristic abnormalities as hypervolemia or behavioral patterns such as physical inactivity.[75] But in the studies described, "independence" was from total cholesterol levels; was the strong atherogenic influence levied by the almost invariable depression of high-density lipoproteins accounted for?

As mentioned, a recent paper refuted the independent impact of obesity on atherogenesis.[8]

More specifically, a component of the Framingham Study has revealed that the 26-year-incidence of cardiovascular disease, in relation to relative (to Metropolitan Life Insurance Company "desirable" values) weight at entry of subjects younger than 50 years of age, was as follows: for men, at relative weight less than 110, 125/1 000 subjects, weight 110-129, 200/1 000 and weight 130+, 267/1 000.[75] For women, at relative weight less than 110, the incidence of cardiovascular disease was 105/1 000, weight 110-129, 121/1 000, and weight 130+, 128/1 000. All these subjects were normotensive, had total serum cholesterol levels less than 250 mg/dl (6.48 mmol/l) (38.6 mg/dl ~ 1 mmol/l), had no evidence of glucose intolerance, and did not smoke cigarettes.[75]

(c) Distribution of Adiposity, and Relationship to Differential Effects on Cardiovascular Risk Factors in Men as Compared to Women

Certain cardiovascular risk factors are more commonly associated with obesity featured by disproportionate accumulation of fat tissue in a "truncal" or "abdominal" distribution.[7,76-78] More precisely, an increased "waist/hip circumference ratio" or "waist/hip girth ratio" (WHR) bears this more common correlation.[7,76-78] The associated risk factors include systolic and diastolic hypertension, and relative glucose intolerance due to insulin resistance, as reflected by positive correlation with total integrated plasma glucose and insulin responses to a glucose load.

The effects of regional distribution of adipose tissue may be independent of, and additive to, those of extent of obesity.[79,80] In fact, within groups of obese subjects (e.g. those 130% of reference weight), relative weight was not predictive, but the "waist/hip circumference ratio" remained a significant predictor of impaired glucose tolerance and elevated triglyceride levels.[79] In addition, while at comparable degrees of obesity, men have higher blood pressure, insulin resistance and triglyceride concentrations, women with "abdominal" obesity have a "male risk profile", in contrast to women with the more usual "peripheral" adiposity.[76] "Abdominal" obesity in (nonhirsute), premenopausal women is associated with, and may be due in part to, increased "relative androgenicity", as reflected by depressed plasma sex steroid-hormone-binding globulin binding capacity, and an elevated fraction of free testosterone.[81]

Why should particular adipose tissue distributions have metabolic sequelae? The answer relates to the regional specialization of fat depots. In women, for example, different regions of subcutaneous adipose tissue, e.g. abdominal compared to femoral, have dissimilar number and affinity of insulin receptors, as well as disparate degrees of post-receptor activity.[81] In obese women with usual fat distribution, higher post-receptor activity may account for enhanced insulin-induced glucose utilization in femoral adipose tissue, probably contributing to greater accumulation in this region than over the abdomen.[81] These more common regional metabolic variations are probably altered in women with high "waist/hip circumference ratio".

It has been suggested that treatment may be indicated for "waist/hip ratios" above 1.0 for men and above 0.8 for women.[7] More specifically, the 12- to 13-year Gothenburg, Sweden study[82,83] has revealed the following: expressing the results as quintiles of "waist/hip circumference ratio", for <u>men</u>, quintiles I (lowest ratio) to V (corresponding to body mass indices 22.2-27.7 kg/m^2) were associated with an increase in systolic blood pressure from 140 to 147, rise in diastolic blood pressure from 86 to 94, and total serum cholesterol from 6.83 to 7.13 mmol/l; except for serum cholesterol, all changes were significant at $P < 0.001$.[82] For <u>women</u>, in groups of quintiles I to V, corresponding to body mass indices 22.5 to 27.0, systolic blood pressure increased from 130 to 142, total serum cholesterol from 6.70 to 7.16, and blood glucose from 4.02 to 4.41 mmol/l, all these parameters changing significantly.[83]

In view of the preceding discussion, the "waist/hip circumference ratio" may be a better predictor of cardiovascular complications and related deaths than other measures of adiposity.

In addition to subjects with "truncal" or abdominal obesity, a less common subgroup is comprised of subjects with massive (hypercellular) obesity.[1] This short digression will illustrate the singular characteristics of massive

corpulence. While massively obese persons, particularly those with "super-massive obesity" (body weights higher than 160 kg, body mass index ~ 40), suffer from devastating early morbidity and mortality, these are frequently due to pulmonary, traumatic and psycho-social problems.[1] In contrast to persons with "truncal" obesity, and contrary to common belief, some massively corpulent subjects, who characteristically have diffuse adiposity, may actually have lesser association with certain cardiovascular risk factors, and a decreased incidence of related complications, than lean subjects.[1] For example, the incidence of atherosclerotic coronary heart and peripheral vascular disease might be even less than in some persons of "desirable" weight. This favorable circumstance appears to apply to both women and men.

(d) Impact of Obesity as Related to Age

As already mentioned, and as also pertains to elevations of total serum cholesterol, the cardiovascular burden of obesity lessens after 45 years of age, and practically disappears after age 60.[1,4,10,11]

II. CONCLUDING REMARKS

At present, it is not possible to provide quantitative estimates of the impact of obesity on cardiovascular risk factors. As discussed, this statement is based on the fact that obese persons do not represent a homogeneous population; rather, a number of sub-groups, each differing in clinical manifestations and complications, comprise the "obesity syndrome". Relatedly, and paradoxically in terms of general beliefs, massively obese subjects, particularly those with unusually high degrees of corpulence, while vulnerable to severe morbidity for several reasons, not uncommonly have relatively favorable risk factor "profiles" relevant to atherogenesis.

Persons with "truncal" obesity, who are not usually massively corpulent, have the strongest association with cardiovascular risk factors. In general, progressively greater degrees and duration of "truncal" obesity have increasingly more pronounced association. However, this positive relationship is by no means linear because a variety of genetic and environmental factors interact to modify the expression and impact of the cardiovascular risk factors. The genetic factors vary of course from family to family.

Much more research is required to enable quantification of the impact of obesity on cardiovascular risk factors, and of the precise role of interactive processes, in each sub-group of obesity.

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